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PULMONARY OVER-EXPANSION IN INFANCY AND EARLY CHILDHOOD[1]

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THE term "over-expansion," used in the title, may be questionable, since by definition the word "emphysema" applies to temporary distention of the alveoli by air, as well as to a permanent one. It is chosen to avoid any implication that the condition is necessarily more than temporary.

The chests of adults and older children are customarily roentgenographed in fullest comfortable inspiration, the lung containing nearly its full normal amount of complemental air. A significant increase or decrease of air beyond this point entails a loss of detail. The usual roentgenograms of infants and small children reveal lesser expansion and aeration than those of older patients, for several reasons.

Most infants' chests are probably examined in the supine position. This is quick, least disturbing to the patient, who is often very weak, and requires no especial fixation device. In this position, the large liver and ever present intestinal gas combine to elevate the diaphragm. The technician tries so to handle the child that it shall lie quietly in a true anteroposterior position. The least movement is apt to affect the position of the thorax, distorting the lung-fields and the important cardiomediastinal silhouette. She hopes to manage with minimum restraint, because restraint is apt to result in a resentful, squirming infant, difficult to maintain in proper position. When she succeeds, the child is usually breathing quietly and will be caught in or at the top of tidal air. The technician may be tempted to utilize the more obvious expiratory pause, in which case the film will be secured below tidal air.

The effect of this can be seen by citing the conventional figures for the divisions of pulmonary air in the adult:

Residual air	1,500 c.c.
Supplemental air	1,500 c.c.
Tidal air	500 c.c.
Complemental air	2,000 c.c.

While it is no longer agreed that the pleural negative pressure has an important rôle in the mechanics of respiration, it is accepted as the expression of the elastic pull of the lung against the thoracic cage (2). It is held that this negative pressure is low or absent in the infant because the volume of the lung at expiratory rest almost equals that of the thorax. This should result in a volume of residual air proportionally lower than in the adult.

[1] Presented before the Radiological Society of North America at the Twenty-fifth Annual Meeting, at Atlanta, Dec. 11–15, 1939.

These factors so combine that the average roentgenogram of the infant's chest has its pulmonary detail depicted on a background of definitely less air than is obtained in the adult. The film tends to resemble that of an older patient taken in expiration. The vascular shadows are rather faint, the diaphragm high, the mediastinum broad, the hila partially obscured, the heart relatively transverse.

Frequently we see films of which the broncho-vascular detail has all the sharpness to which we are accustomed in the adult. The diaphragm is depressed, the heart vertical and slenderized, the mediastinum narrow, the hila slender but well uncovered, the ribs elevated, and the intercostal spaces broad. The whole configuration of the chest approaches that of the adult.

This may be normal. A young child may have co-operated for a full inspiration. An infant may have been caught at full inspiration, particularly while crying vigorously. Pancoast's technic (12), in examination for enlarged thymus, of securing an accurate inspiratory view in the upright position, while the child is crying, regularly may give this picture. As he stated, this requires painstaking care. This inspiratory view is of interest in the light of his comment that the large thymus is not necessarily broad, but may be narrow and deep.

Devices are used to fix the infant in the upright position for examination. These secure better aeration of the lungs and better information concerning the thymus and aorta and position of the trachea (13, 15). Inspiratory films so secured depict a degree of aeration which would indicate a real over-expansion if obtained in the supine position in quiet breathing.

On the other hand, there appears to be a definite advantage in having routine films taken in the supine position at quiet inspiration, accepting the lower aeration, broader mediastinum, and obscured hila, and discarding those films which appear to be frankly expiratory. This has seemed to give a ready comparison with cases of over-expansion and has seemed particularly useful in recognizing those in which the lung was persistently maintained in a state of mild over-aeration. Such evidence must be accepted cautiously, due often only to its persistence on re-examination and only as supported by clinical facts.

The variability of aeration in the normal infant may be inferred from the investigations of Deming and Washburn (5). They found that with the infant awake and quiet, the pulmonary ventilation varied from 555.8 to 3,213.7 c.c. of air per minute, averaging 1,319. Crying, it varied from 1,551.4 to 5,943.9 c.c. per minute, averaging, 3,535.3.

Allen and Waltz (1) have listed the roentgen signs of the "inspiratory state," and have given a useful index of its degree. This is the ratio of the distance between the inner end of the clavicle and the middle point of the collateral dome of the diaphragm to the width of the thorax. This, they find, is approximately 0.45 to 0.53 in full expiration and from 0.6 to 0.75 in full inspiration. The younger the infant, the lower is this index. The index can be impaired by the particular type of thoracic expansion. Exceptional increase in the width of the thorax in inspiration has at times nullified the effect of increased length, so that over-expansion may occur with a normal index. It has, however, proved to be easily applied and generally reliable. Unless means are taken to insure full inspiration routinely, the inspiratory state, particularly if exaggerated, will probably be seen more often as a persistently maintained expansion from disease than as a normal full inspiration. Such over-expansion contrasts sharply with the moderate aeration of the usual supine film made in quiet inspiration. It may be local or general and is seen in a wide variety of conditions.

There are several mechanisms by which over-expansion is produced. The one most frequently cited is that of partial expiratory obstruction. Because of foreign body, bronchial stenosis, or viscid bron-

chial secretions, air which easily enters is not readily expired. This results in air-trapping, which may be either general or local.

Deficient pulmonary ventilation may cause compensatory over-breathing and over-expansion in the absence of mechanical obstruction or of primary pulmonary disease. This may occur in congenital cardiac disease or pulmonary edema.

Compensatory emphysema occurs whenever a significant amount of lung loses ventilating power. This arises from many diseases, such as pneumonia, atelectasis, tuberculosis, pneumothorax, congenital cystic disease, bronchiectasis, or pleural effusion. The disease may cause pathologic enlargement of the involved regions, as in congenital cystic disease, bronchiectasis, or chronic emphysema. To this enlargement is added a compensatory over-expansion of the functioning portions of the lung.

Resistance to respiration (inspiratory and expiratory) has been experimentally studied. These studies cite the effects on efficiency of ventilation, as to carbon dioxide elimination and oxygen absorption, and as to the total ventilation volume of air (9). Little is said concerning the effect of this resistance on the air permanently retained in the lung, except in relation to expiratory resistance or "air-trapping." Our films give us reason to believe that even in inspiratory resistance, particularly in the infant and small child, the permanently retained air tends to be high. This has been seen in acute laryngeo-tracheo-bronchitis, before relief by tracheotomy.

Killick (9) has shown experimentally that increasing inspiratory resistance is met first by full ventilation, a heightened carbon dioxide alveolar tension, and good carbon dioxide elimination and oxygen absorption. Progressively, carbon dioxide elimination and oxygen absorption fall (the former more rapidly), the respiratory center and muscles become fatigued, and ventilation volume decreases. From the film it appears that permanently retained air in the lungs falls rather late in respiratory failure in the infant, except as it is lost in atelectatic or consolidated lung. If there is a normally low residual air in the infant, there would be especial need to maintain a high level of expiratory aeration in threatened respiratory failure, to minimize the deficiency of gaseous exchange and to prevent atelectasis.

The most common cause of infantile pulmonary hyperexpansion is acute respiratory infection (16). The commonly accepted mechanism is expiratory resistance by viscid bronchial secretions. This may occur in involvements of all degrees of severity, from mild tracheobronchitis to severe fatal pneumonia (Fig. 1). The former particularly may arise surprisingly soon after birth, with few signs beyond those of respiratory difficulty (Fig. 2). At one time, at least half of our examinations for enlarged thymus yielded typical over-expansion films, frequently of exaggerated type, which contrasted sharply with the classical picture of thymus enlargement. So brief and mild are the signs of infection in some respiratory attacks that both the attack and associated hyperexpansion would be difficult to credit, if autopsy were not occasionally obtained because of fatal sequelæ of mastoiditis or intracranial infection.

First films may reveal over-aerated, apparently clear lungs, later ones showing gradually progressive or shifting infiltrations of pneumonia and atelectasis. The earlier emphysema may be lost or obscured. The earlier stages of pneumonia may be difficult to recognize on the background of highly aerated lung. Kerley (8) emphasizes that pneumonia in the small infant tends to localize at the paravertebral regions, where it is additionally obscured by location. At times small pneumonic zones appear so stretched by surrounding over-expanded lung that they resemble the band-atelectases which have been reported in the chests of adults. Many writers have commented on the tendency of atelectatic zones to lie dorsally in dependent locations, while the ventral regions have air-trapping and attendant emphysema.

Evidence of over-aeration may be obtained incidentally in severe non-respiratory disease which so dominates the clinical picture that an intercurrent or terminal respiratory infection is not recognized. One infant dying with small intestinal volvulus, without evident respiratory symptoms, at autopsy was found to have emphysema and bronchial secretion. Over-expansion of the lungs was incidentally shown on the film of the abdomen.

In acute laryngotracheitis, with severe laryngeal stenosis and inspiratory dyspnea, there may be marked hyperexpansion. This can occur with the marked inspiratory retractions of the thorax which have been described by Jackson (7). (See Fig. 3.)

Case 1. W. H., Caucasian male, aged 19 months, was admitted in a state of severe inspiratory dyspnea with laryngeal stridor which had arisen suddenly. He had done well after being discharged as convalescent from pneumonia ten days previously.

The tonsils were moderately enlarged, the oro-pharynx slightly infected. The ala nasi were active. There was retraction of the lower ribs and sternum on inspiration. The chest was resonant, the breath sounds and any râles were obscured by the stridor. Temperature was 101.8°.

Culture from oral secretions revealed no Klebs-Loeffler bacilli. Relief followed emergency tracheotomy. The fever dropped slowly by lysis, with occasional exacerbations. The child was discharged as asymptomatic after 41 days.

The film before tracheotomy (Fig. 3) revealed a definite inspiratory state, with an index of 0.69, definitely above that usually obtained in our films, and well above that shown during the patient's previous admissions for pneumonia. There was diffuse infiltration in the right upper lobe and scattered patchy foci. While the mediastinum was somewhat slenderized by the inspiratory state, it was wider than normal. Edema may have been responsible.

The mechanism of the inspiratory state

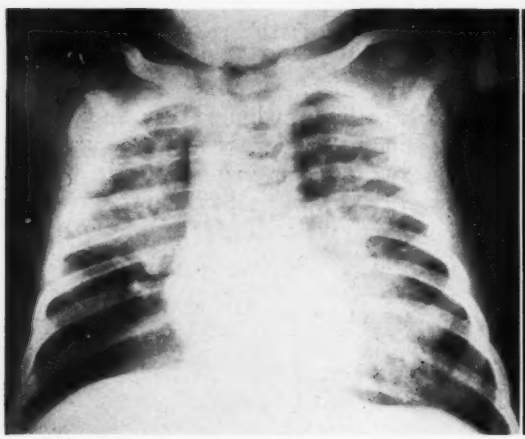

Fig. 1.

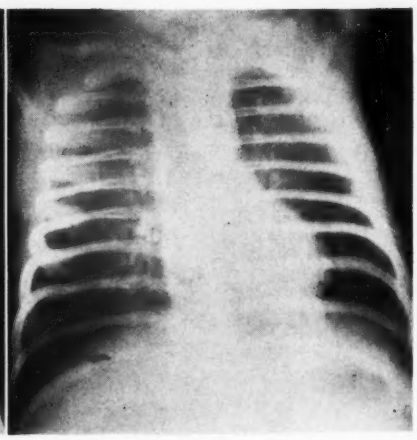

Fig. 2.

Fig. 1. Caucasian female, aged 4 months. At autopsy, there was extensive bilateral bronchopneumonia. The tracheobronchial tree was filled with purulent material, its mucosa was injected and hyperemic, and exudate adhered in patches. Over-expansion, inspiratory index 0.7.

Fig. 2. Caucasian male, aged 6 days, with hare-lip and cleft palate. Surgical reconstruction was delayed and the chest radiographed because respiratory difficulty roused suspicion of enlarged thymus. Temperature averaged 99.5 for a few days at the time of examination, otherwise was subnormal. The respiratory difficulty subsided quickly, and surgery was done later without incident. Over-expansion from a mild, transient respiratory inflammation. Inspiratory index 0.79.

in the case rests between secretion over-expansion and that secondary to inspiratory dyspnea. The child was maintaining a high air content against an inspiratory resistance sufficient to require emergency relief.

Peirce and Dirkse (14) have described the production of pneumatocele (local or lobular ectasia) by local bronchial obstruction in respiratory infection (Fig. 4). These may remain permanently, to simulate congenital cysts, or may disappear with subsidence of the infection. Since studying the work of these workers, we have not infrequently seen these apparent cavities disappear with resolution of the accompanying pneumonia. It is well to allow time for this before diagnosing tuberculosis, abscess, or congenital cyst.

Pulmonary tuberculosis in infancy and early childhood shows no especial tendency to over-expansion. When pneumonia and tuberculosis with parenchymal spread are to be differentiated, the presence of emphysema and absence of visibly enlarged tracheobronchial glands are definite points against the latter. Large tuberculous glands frequently produce enough bronchostenosis to cause emphysema, which may be local or general according to the location and extent of the stenosis (6, 17). (See Fig. 5.) At times narrowed bronchi may be sharply depicted on the background of glandular masses or parenchymal consolidation. The mediastinal shadow is broadened irregularly by the glandular enlargement, but this may be made less conspicuous by the effects of the emphysema. Progress examinations may be required to determine whether over-expansion is due to intercurrent infection or to glandular pressure.

Miliary bronchopneumonia is occasionally seen as a sequel of measles. Supposed miliary tuberculosis, after this disease, must be diagnosed with caution, particularly if the visible tubercles are sparse, hazy, and coarse, are without glandular mass, and are associated with high aeration of the lung. Progress examinations are needed to complete the diagnosis.

Bronchiectasis in the young varies greatly in extent and roentgen manifestations. Anspach (3) has described a common form arising from atelectasis of a lower lobe, and mentions hyperexpansion of lung adjacent to the atelectasis. There is a more widespread type in which fusiform dilation of terminal bronchi is extensive, and may involve all lobes. We have seen

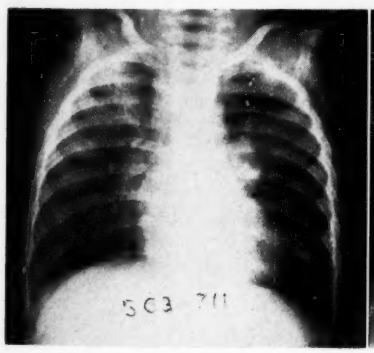

Fig. 3.

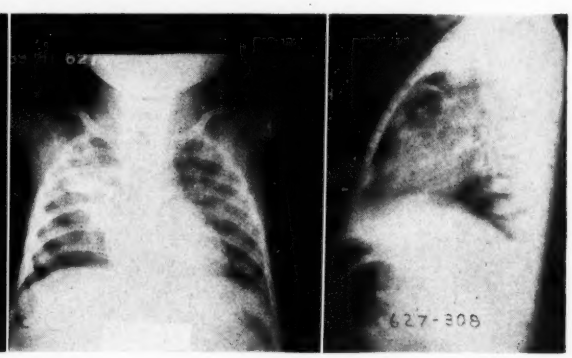

Fig. 4.

Fig. 3. Caucasian male, aged 19 months, with acute laryngotracheitis, requiring tracheotomy. In spite of severe inspiratory dyspnea, the chest was over-expanded; index 0.69. There was associated pneumonia. The inspiratory state was higher at this time than in the same patient on previous admissions for pneumonia without laryngeal complication.

Fig. 4. Caucasian female, aged 9 months, was admitted with acute respiratory infection consistent with pneumonia. Tuberculin reaction persistently negative. Sputum contained pneumococcus, type 7. Became afebrile after sulphapyridin and made good recovery; the cavity disappeared. Local pneumatocele, which disappeared after resolution of pneumonia.

this associated with marked general over-expansion and patchy, widespread, pneumonia-like foci on the films, and with evidence of emphysema at autopsy. These have been without the triangular basal density noted by Anspach, or evidence at autopsy of origin in basal atelectasis (Fig. 6). Such involvement may be congenital in origin, but it would appear best to be conservative in this conclusion (3, 10). In these cases over-aeration compensatory to much non-functioning lung, added volume from dilated bronchi, expiratory resistance from much bronchial secretion and secondary organic emphysema, appear to combine to give marked over-expansion.

Congenital cystic disease, if sufficiently extensive, may cause hyperexpansion, both by the volume of its cystic areas and by the compensatory emphysema induced in the functioning portions of the lungs. The cystic areas will usually be evident if the involvement is extensive enough to be significant. Intercurrent infection may exaggerate the picture of over-expansion, or by superimposed infiltration may cause the cysts to resemble the cavities of tuberculosis or abscess (10).

Lipoid pneumonia, unless produced by the more irritating oils, does not appear to be of a pathologic type calculated to produce over-aeration primarily. From the cases we have seen, it appears the over-expansion in this condition is caused by massive lesions, which incapacitate sufficient lung to induce compensatory over-aeration, or to be due to intercurrent respiratory infection. The distribution of the infiltration in the perihilar and median dependent regions of the lungs, more marked on the right side, is described by Bromer and Wolman (4), and may assist in its recognition. Its prevalence in the constitutionally weak infant is important. Perhaps the absence of material over-expansion with scant pneumonia-like infiltrates under appropriate circumstances may be a diagnostic point for the disease (Fig. 7).

Congenital cardiac diseases are fre-

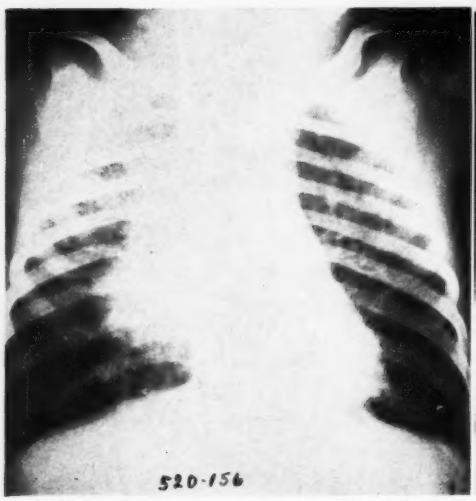

Fig. 5.

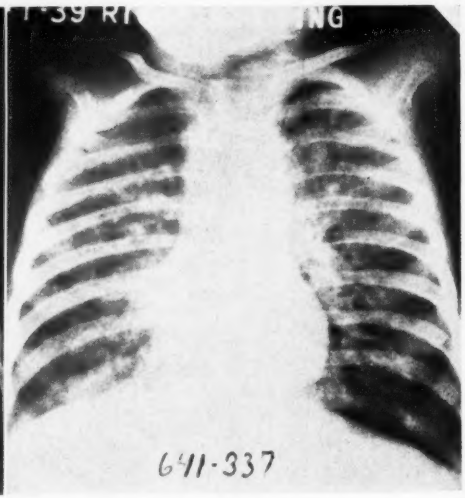

Fig. 6.

Fig. 5. Ethiopian female, aged 2 years, whose mother was thought to have inactive tuberculosis, was admitted with one of several episodes of wheezing respiration, noisy râles, and rhonchi. The tuberculin test was positive. Films over a period of 10 months do not differ materially from this. Tuberculosis with over-expansion; index 0.7. Enlarged tracheobronchial glands and parenchymal involvement.

Fig. 6. Caucasian male, aged 1 year. At autopsy, four days after this film was made, all peripheral bronchi were found to be markedly dilated. Foci of pneumonia and of abscess were numerous about the peripheral bronchi. Widespread bronchiectasis with focal pneumonia and abscess. Over-expansion with an index of 0.72.

quently associated with pulmonary hyperaeration. They are apt to require a heightened pulmonary ventilation to insure sufficient gaseous exchange. Mixed systemic blood appears calculated to stimulate the respiratory centers. In addition, the thorax may be given a spurious impression of over-expansion because of the increased volume required for an enlarged heart. The heart may cause compression atelectasis, producing the median-basal triangular shadow of Anspach (3).

Case 2. R. C., Caucasian female, was blue at birth but cried lustily. Breathing was always rapid. At five weeks of age, she suddenly developed a gasping respiration and was admitted to the hospital: temperature 97.4, pulse 134, respiration 54. There was purulent nasal discharge and slight infection of the throat. Inspiration was loud and short, expiration grunting; the chest moved symmetrically. There was inspiratory supraclavicular and sternal retraction. The cardiac rhythm was rapid and regular, the tones were good, without murmurs. Over the left lower lobe were diminished resonance and breath sounds, and fine to medium moist râles. There was general cyanotic mottling of skin, but no edema of the extremities. Hemoglobin, 88 per cent; red blood cells, 4,600,000; white blood cells, 23,000; neutrophiles, 64 per cent; lymphocytes, 32 per cent. Wassermann-Kahn reactions were negative.

The provisional first diagnostic impressions included congenital cardiac disease, enlarged thymus, atelectasis, laryngeal stenosis, complicating bronchopneumonia.

Autopsy two days after entry revealed: A large heart with numerous transpositions of great vessels, whose main features include venæ cavæ entering the right auricle, the aortic arch leaving the right ventricle and carrying the upper great vessels; the descending aorta leaving the left ventricle, carrying its normal branches and the pulmonary arteries. Joining these separately arising aortæ was a zone of stenotic aorta and a patent ductus. Pulmonary veins entered the left auricle. The interventricular septum has a defect 3 mm. in diameter. The interatrial septum is intact. The essentials of this, for present purposes, appears to be a veno-arterial shunt, selectively severe in the blood supply of the respiratory center. The lungs revealed extensive dependent bilateral atelectasis which must have progressed markedly in the day before death. No pneumonic areas were identified. The bronchi were compressed but without exudate.

The over-expansion of the lung is obvious on the film (Fig. 8). The inspiratory index is 0.85.

There is obvious cause for over-breathing and none for expiratory obstruction. The inspiratory retractions and the degree of aeration indicate that the child was carrying inspiration to the limit of the expansibility of her lungs. Atelectasis was be-

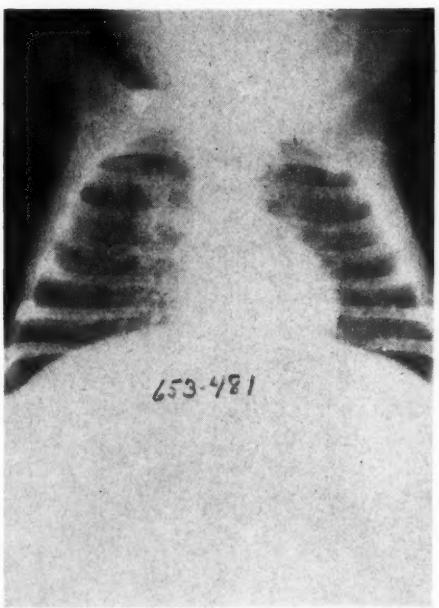

Fig. 7. Caucasian female, aged 4 months. Autopsy, one day after this film was made, revealed a Mongoloid infant with malnutrition and dehydration. Lipoid pneumonia was scattered in small patches in the upper lobes. The film shows questionable over-expansion, index 0.6. The involvement appears not extensive enough to cause material over-aeration. Massive involvements have shown it definitely.

ginning. The respiratory centers must have been on the verge of final failure.

Transposition anomalies like the above appear usually to be associated with a globular heart and a narrow mediastinal shadow. The latter heightens the impression of over-expansion.

Hyperexpansion of lungs was encountered in a child suffering from rabies.

Case 3. P. N. G., Caucasian female, aged 5 years, admitted with advanced rabies following the bite of a puppy three weeks before entry. Temperature 104.6, pulse 200(?), respiration 40(?). There was cyanosis of the lips and skin, maniacal symptoms developed on the slightest irritation, with staring eyes, bilateral strabismus, facial grimaces, gnashing of teeth, laryngeal spasms, and spasms of muscles of deglutition. There was a peculiar, shrill cry. A film was taken the day before death.

At autopsy, impression smears showed Negri bodies in the hippocampal gyri. The right lung weighed 150 grams, the left 140; both were soft, fluffy, and crepitant throughout. Along the free margins of the left upper lobe were several small emphysematous bullæ, associated with a few petechial hemorrhages in the pleura. On section, the lungs had a pinkish-gray color, and a slight amount of edema fluid was present. The bronchial mucosa was not injected and the bronchi contained a frothy, serous exudate. Tracheobronchial lymph nodes were not enlarged (Fig. 9).

The lungs showed an inspiratory state not beyond that obtainable from a fully co-operative child five years old. Since the film had to be secured in a quiescent period, the picture is one of definite over-expansion. The emphysematous bullæ may or may not have been due to trauma of the disease, although associated with petechial hemorrhages. There were no viscid secretions. Edema may have been sufficient to induce some over-expansion; laryngeal and bronchial spasm may have been a fac-

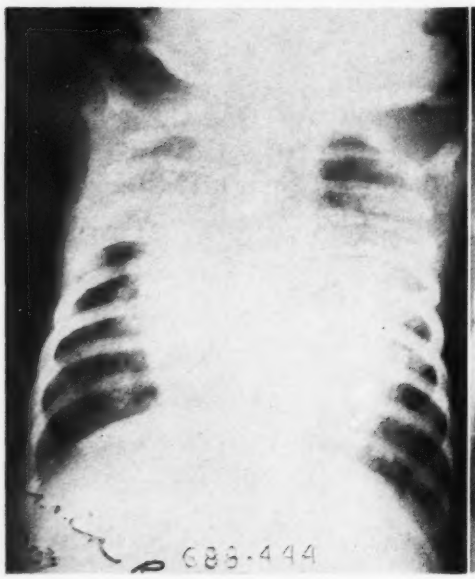

Fig. 8.

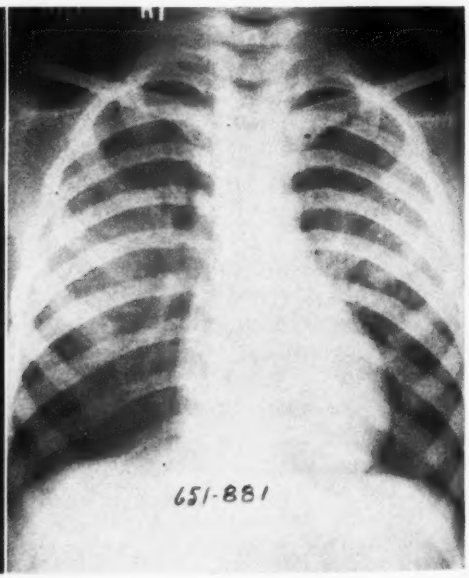

Fig. 9.

Fig. 8. Caucasian female, aged 1 month. Congenital cardiac disease. Autopsy revealed a complex transposition of the great vessels, and atelectasis. Marked over-expansion, index 0.85.

Fig. 9. Caucasian female, aged 5 years. Died of rabies on the day of examination. Autopsy revealed emphysema and petechial hemorrhages. Moderate over-aeration, index 0.74.

tor. Since the over-aeration was not marked enough to exceed the elastic limit of the lung, it would not be expected to appear at autopsy.

COMMENT

Pulmonary over-expansion in the infant and small child is an entirely non-specific condition caused by various mechanisms. It may be simulated by a chance, very full inspiration. If due to actual disease, it may be a temporary over-aeration in which no recognizable injury is done to the alveoli. Correspondingly, it may or may not be evident at autopsy, depending on whether or not the elastic limit of the lung is exceeded. In any given case, it is to be judged in relation to clinical and roentgen manifestations.

As infancy advances into childhood, it is less frequently seen, partly because the films are taken in fuller inspiration, partly because a temporary abnormally heightened aeration less frequently occurs.

Used with due caution, the manifestation is frequently of interest and sometimes of diagnostic value.

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DISCUSSION

GEORGE WYATT, M.D. (Boston): I appreciate the privilege of discussing Dr. Carter's paper. I am glad he has called attention to the fact that there is a greater difference between adult chests and infant chests than that of size. In addition to the variations in technic, the pathologic changes that occur in infant chests have different manifestations. When viscid bronchial secretions are present, the manifestations of obstruction are naturally more pronounced in infants and small children than in adults, the bronchial lumena of whom are relatively larger.

The most frequent cause of pulmonary over-expansion at the Children's Hospital in Boston, as judged by those cases that come to postmortem examination, is bronchial obstruction. In the majority of cases, this obstruction is due to the viscid mucopurulent secretions of interstitial pneumonia.

I am glad that Dr. Carter mentioned the thymus. Dr. Wasson, in his refresher course on the chest at this meeting, also discussed the thymus. Both Dr. Carter and Dr. Wasson hold the opinion that a large thymus, or the exposure of a large

thymus on an x-ray film, is of very little significance as far as the child's prognosis and respiration are concerned. We have found the same thing in Boston. Dr. Farber, Pathologist of the Children's Hospital, states that in 3,000 autopsies he has yet to see a single case in which he could attribute the death of the infant to a large thymus.

I would like to say a word about technic, because that is one of the most difficult problems with which we have to deal in infants. In adults, it is possible to obtain comparative films because the patients can usually take about the same depth of inspiration. Thus consecutive films may be made on the same patient and comparative films on different patients. In infants, there is no such co-operation. Infants, however, have one thing in common. If they are immobilized—as is usually necessary in order to get good films—they become righteously indignant and usually cry vociferously and, in most cases, rhythmically. It is not necessary that they cry rhythmically, but cry they will if they are strong enough. The cry is nearly always of the same type, namely, a rapid inspiration and a prolonged, slow expiration with the cry. We have found that in order to obtain comparative films on these infant chests the exposure should be made at the beginning of expiration, and the infant should be held by two attendants. One holds the head between the arms and the other holds the legs. The infant becomes angry and cries, and the technician presses the button at the beginning of an expiration.

We have found it impossible to get these films at the height of inspiration because the height of inspiration is too transient. The diaphragm comes down and immediately starts up again, but although the diaphragm is moving when the film is snapped, if the infant is crying and the egress of air is impeded by the larynx, the movement of that diaphragm is so slow that motion is not a highly deterrent factor.

We take these films, using an impulse timer at 120th of a second, which gives an effective exposure time of about 250th of a second.

ROBERT R. NEWELL, M.D. (San Francisco): I found Dr. Carter's exposition very instructive indeed, and I think perhaps he has taught me something that will help me further in future work with babies' chests. The discussion already has covered some of the things which I have been planning to say.

However, I have been puzzling, ever since Dr. Carter began, to analyze or to figure out and get in an orderly way the fundamentals which would explain the differences that we experience between babies' chests and adult chests.

Unfortunately for most of us, our ideas in regard to chest diagnoses are made up very largely on an experience with adult chests. It is true that the baby's metabolic rate is perhaps twice as great per pound as it is for the adult, so that there would be needed a greater porportional aeration. But when you increase the size of a body you increase the volume at a greater rate than you do the surface, so that the surface goes further, perhaps, in a baby than it does in the adult. The result is that there is more room in the baby's chest for interstitial tissue—and still have an adequate surface in the air cells.

Also, there is a shorter distance required for diffusion, for movement of the molecules in diffusion to get from the air vesicles out into the larger bronchi so that the baby again can get along with perhaps a somewhat smaller surface than would be expected. All those things let the baby get along with more interstitial tissue in the lungs and less actual space in the air vesicles. The air vesicles in the baby are smaller than they are in the adult. The result is less contrast.

You would think that you would get a brighter contrast in a baby's chest because you always take the film with a lower kilovoltage. That is true for bones but it is not true for vascular and soft tissue shadows. It is easily demonstrated that

the contrast of soft tissue is very little different for 40 kilovolts compared to 80 kilovolts, whereas the difference for bones is very large for that difference in kilovoltage. You do not get the brighter contrast that you might expect in a baby's chest when you lower the kilovoltage.

I was very much impressed by the films that Dr. Carter showed and do think that most of the over-aeration is due, as was suggested by the discussant, to partial obstruction in the bronchial drainage. The baby's bronchi are small and it does not take very much swelling of mucous membrane and thick mucus to plug them up to a degree which would not happen in an adult chest.

It is easy to show that a partial obstruction will naturally result in air-trapping behind it, because of the powerful muscles that fill the lungs. Ordinarily, in quiet respiration, the lungs are emptied by elastic recoil, which is a small force. So that partial obstruction always results in filling up of the lung, and it takes a complete obstruction to result in atelectasis.

RAY A. CARTER, M.D. (*closing*): I am glad that Dr. Wyatt emphasized matters of technic, which I could not cover adequately. Proper technic is important. Films taken in the supine position with quiet breathing are not always adequate. Special technics are frequently called for.

Dr. Newell's comments explain factors which I had not realized in the production of the appearances seen in the infant's chest, and I am glad to have them stressed.

Over-expansion of the infant's chest obscures much real pneumonia, atelectasis, and other pathology. Kerley has emphasized this, particularly that the medial location of bronchopneumonia in a small infant conceals it behind the heart and mediastinum.

QUALITY OF ROENTGEN RAYS: EFFECT ON TISSUE DOSE, AND CLINICAL IMPORTANCE[1]

By R. R. NEWELL, M.D., *San Francisco*

From the Stanford University School of Medicine

HIGH voltage x-rays are used mostly for treating cancer, and that will be the tacit principle underlying these remarks.

For ten years I have been thinking and talking about the development of harder qualities of x-ray for therapy. In 1930, half a million volts was the limit; to-day several are treating at a million and the use of two million is imminent. The next step will take us clear beyond the gamma-ray region.

In that decade much has been learned about absorption of x-rays and their biologic effects; and the whole field of nuclear chemistry has been opened to exploration. The neutron, positron, and maybe three other elementary particles have been added to the two previously known, and matter and radiation have been observed turning one into the other. It has been a time of truly revolutionary development in physical knowledge. Yet, already, in 1930, we knew enough about x-rays to make a fair prediction that the cancer problem would likely not be solved by application of a million volts.

The physical aspect of irradiation of a patient is of energy released within the part irradiated. This has two faces, namely, the gross distribution and the fine distribution. The former we call "depth dose" and "surface dose." As we go from very soft qualities to very hard ones, we find depth dose increasing. The increase is exceedingly rapid at first, for at long wave lengths the absorption in flesh is mostly by the photo-electric mechanism, for which the absorption coefficient varies with the third power of the wave length. But as we get into the range of h.v.l. = 1 mm. Cu this has, for light elements, fallen to a level where it is of less importance than the true absorption of scattering,[2] for which there is little change with wave length. From here on, the depth dose gains but slowly. There are some secondary effects—lessened backward and lateral scattering with increased amount of scattering in a forward direction—which favor extremely short wave lengths for treating deep tissues of a large patient. But the reward in increased percentage depth dose is pretty small for the large increase in cost.

The mechanism of scattering, which dominates the picture for hard qualities, results in lengthening of wave length (1/20 Ångström for x-rays scattered directly backward). Depth dose, being composed partly of scattered ray, largely so for great depths and large fields, is, consequently, a good deal softened, however hard the incident beam. To use an analogy: one buys a clear blue light "of purest ray serene," and finds, to one's horror, that the room is flooded with purple. Clarkson and Mayneord (2), on the basis of measurements with a differential ionization chamber, give a detailed picture of the quality in the depth for various qualities incident on different sized fields. Payne-Scott (3) has calculated the relative importance of the primary and scattered x-rays in the depth. After studying these, one begins to appreciate the enormous importance of scattering for clinical roentgen therapy. Apparently ordinary deep therapy (h.v.l. = 1 to 2 mm. Cu) is in the region where scattering is relatively the most important. But scattering does not make all incident qualities look alike in the depth—what starts harder will end up harder.

[1] Read at the Third International Cancer Congress, in Atlantic City, N. J., Sept. 11–16, 1939.

[2] Trueblood and Loughridge (1) have studied the picture of the absorption of 800 kv. x-rays in nitrogen. It is very complicated. Their finding of fifty times the theoretical number of photo-electrons still does not alter the above argument.

The first consideration, however, is quantity in the depth. For deep-seated tumors one needs a high depth dose (expressed as percentage of the tissue dose at the surface). Large portals, irradiating large volumes, increase depth dose by increased total scattering. But there is a limit to this. Harder qualities penetrate deeper, but beyond h.v.l. = 1 mm. Cu only a little deeper.

All those who have carried measurements of depth dose into the supervoltage range agree that it shows discouragingly slow improvement at great expense of cost and effort. One gains in depth dose as well by increasing the distance from tube to patient as by adding thick filter, and at less cost of increased treatment time. Quimby (4) has discussed the economics of this.

Stone and Aebersold (5) have shown how modest is the improvement in depth dose even on going to 1,000 kv. For cross-fire through opposite ports, supervoltage seems to be worth having only for parts thicker than the neck, and really valuable only for a pelvis larger than average size. In the depth, a superhard beam falls off less toward the edges. Also, and for the same reason, cutting down the size of the beam diminishes the depth dose less for superhard x-ray than for softer qualities. Supervoltage might induce one to plan cross-fire technic with more and smaller ports.

When one records therapeutic x-ray exposures in roentgens, measured in air (of which I am emphatically in favor), one finds that larger doses are needed to produce the same apparent effect (erythema) when one uses harder quality. Lauritsen (6) has calculated the energy absorbed in a millimeter layer at the surface for a given exposure (in roentgens), revealing a large dependence on wave length. Clinically, the change with quality seems to be not nearly so great. But we are working with fairly heterogeneous beams, while he was calculating for monochromatic beams.

But one must not suppose that equal tissue doses of different qualities are actually equal. There is a physical difference between hard and soft irradiation.

This brings us to the second phase of the physical aspect of quality, namely, the fine structure of distribution of energy in tissue dose. Failla (7) has discussed this well. The first step in absorption of x-ray is the production of a high speed electron. This may be a photo-electron containing all the energy of the x-ray quantum which produced it—the shorter the wave length the higher the energy. Even at modest voltages and filters such photo-electrons will have a path in flesh longer than a cell.

Or it may be a recoil electron produced by scattering, under which circumstance it will contain only the difference in energy between the incident ray and the softer scattered ray. This softening is 0.024 Å. for scattering at 90° and cannot be larger than 0.048 Å. (180° scattering). The proportion given up increases as the energy of the x-ray (quantum) increases, so that for $\lambda = 0.05$ the most energetic recoil electron would have almost half as much energy as the possible photo-electron from the same wave length. But for longer wave lengths the recoil electrons get rapidly less energetic.

Within the therapeutic range we have three zones, then, merging one into the other, as follows:

(*a*) Soft x-rays, of the order of 0.5 Å., producing mostly photo-electrons with a range of one or a few cell diameters.

(*b*) "Deep therapy" of the order of 0.15 Å., producing predominantly recoil electrons and these of average range somewhat shorter.

(*c*) Supervoltage beyond 0.05 Å., producing recoil electrons of long path.

Mayneord (8) has tried to evaluate these effects and gives a table of average electron path in terms of cell diameters. This has its importance in the fact that the second step in absorption of x-ray energy is ionization by the high speed electrons. And this ionization is much more intense toward the end of the path. Photographs of these ion trails show them to be very crooked toward their end. The effect on a

living cell is undoubtedly due to this ionization. An erythema dose of x-rays might produce 100,000 pairs of ions in the volume of one cell. This means that the cells are simply riddled by paths of ionization, a thousand or so ions in each path. Yet almost all the cells recover. Does death occur by repeated insult, or is it the chance of a hit in some small vulnerable spot in the nucleus? Many workers, observing the shape of survival curves in biologic tests of roentgen death point, have noted that simple probability of hitting or missing would predict a similar curve. Some have even calculated the size of the vulnerable spot for bacteria. Some have thought they could distinguish between qualities of x-ray which kill a cell if they hit the right spot and other qualities which have to make two hits to kill the cell—for one protozoon, 50 hits to kill. The conclusion is uncertain, however, because chemicals can produce similar survival curves. It has even happened that the survival curves were similar (for a protozoon) whether the culture medium was irradiated before or after the animals were put into it. These experiments on bacteria and protozoa use very large doses—many thousand roentgens. But even in the case of *Drosophila* eggs, in which the dosage is around 200 r, it appears again that x-ray effect may be indirect, by interfering with some developmental organizer at a critical stage of differentiation (9), rather than by killing the cells directly. Muller (11), by studying the effect of summations of x-ray and gamma-ray doses on *Drosophila* pupæ, concluded that their mode of biologic action must be identical.

Yet Henshaw and Francis (12) report a large influence of wave length on relative effect on diverse biologic test objects (seeds and flies' eggs).[3] Quimby and Pack (13) report that combinations of soft with hard rays are less effective in producing skin erythema than any single quality. It is hard to understand these experimental results, in some of which the time-rate of application of energy varied from one quality to another. In others the different test objects were quite dissimilar in susceptibility. One wonders if Love's (14) calculations might have a bearing. He has indicated some extraordinary effects to be anticipated from changing relation of rate of application to the duration of sensitive phase during the cycle of mitosis.

Genetic experiments point strongly to direct action of x-ray on a tiny volume of the cell—perhaps a single biomolecule. Apparently x-rays can alter a single gene within the chromosome and leave the rest of the cell unaffected. Most mutations produced by x-ray can be accounted for perhaps by more diffuse influences on chromosomal migrations during maturation, but some do seem to be point-effects. One must not generalize too broadly from genetic effects, for they are known to differ from most other biologic effects of x-rays in two ways, namely, they are independent of quality (15) and they show zero recovery rate.

The total energy delivered to a cell by a dose of x-ray is quite small indeed. It is delivered to a small linear portion of the cell, and within this track the concentration of energy is high—high enough to meet any demands for doing chemical work. Such a track disappears quickly by recombination of ions. Summation of x-ray dose must be by summation of biologic effect, not by summation of energy, otherwise the effect of time-rate of application would be large. Kingdon (16) found little influence of time-rate over a ratio of 260,000. Some biologic effects may actually be produced by a single "hot spot" of ionization in a sensitive volume.

Kingdon's enormous volume densities of ionization still fell far short of the columnar density along an electron path. Zirkle (17), using the alpha rays of polonium, studied the effect of ion densities a hundred times greater. He found fern spores killed four times as effectively when their nuclei were at the tail of the alpha ray track where the ions are twice as thick as at the beginning of the track.

Neutron rays produce in flesh mostly re-

[3] Henshaw is reporting further at this Congress.

coil protons, with columnar ionization comparable to alpha rays. They are found (18) to be only from two to ten times as effective biologically as x-rays. It is obvious, therefore, that Zirkle's observed high rate of biologic dependence on ion density cannot continue down into the x-ray region (photo-electrons and recoil electrons). Nevertheless there is still the possibility of an important concentration of biologic effectiveness in the densely ionized coiled-up terminus of an electron track. On this view each recoil and photo-electron would be equally effective, and all one would accomplish by going to still higher supervoltages would be to dilute the tissue dose with ineffective, long, sparse ion tracks preliminary to the effective termini. One would give more roentgens for equal effect, but without any perceptible basis for altered differential biologic action between one tissue and another or between cancer and host.

Stone is reporting at this Congress that 200 and 1,000 kv. x-rays can provoke apparently identical cutaneous reactions. A more clean-cut test of the relative biologic action of much sparser columnar densities of ionization could, I suppose, be studied by using exceedingly high voltage cathode rays on quite thin biologic specimens. Haskins is reporting at this Congress some results with low voltage cathode rays.

The opposite, namely, comparison with extremely heavy columnar densities, is already being well studied by means of neutron rays. Biologic differentials have been shown. It remains to be seen whether or not these differentials will work to our advantage in cancer therapy.

CLINICAL IMPRESSIONS

My personal clinical experience concerns three qualities, namely, h.v.l. = 1 or 2 mm. Al, 0.8 mm. Cu, and 5 mm. Cu. On the whole, I am struck with the similarities of roentgen reactions from all these qualities. Stone (19) reports a similar impression.[4] More and more lively does the suspicion become that observed differences of reaction are due less to specific biologic effect of different qualities than to the different ways in which we use different qualities. Rate of application, degree of protraction (fractionation), sizes of areas, and methods of cross-fire are all variables which are hard to allow for in our clinical evaluation. Different parts of the body recover from x-ray injuries very differently. The skin and the mucous membrane develop their roentgen inflammation at different rates—and differently in different parts of the body. The face will bear much more abuse than the hand. The buttock requires a third again the dose to produce desquamation that is needed on the suprapubic region. Different patients are unlike; there are, in fact, idiosyncrasies. And arteriosclerosis damages greatly the patient's ability to recover from roentgen injuries.

All these things make anyone's clinical conclusions open to skepticism. I will venture the following impressions, however:

(*a*) Roentgen "epidermiolysis" looks remarkably alike whether produced by h.v.l. = 1 mm. Al or 5 mm. Cu.

(*b*) We have seen more late roentgen injuries from the intermediate qualities. But we have used h.v.l. = 5 mm. Cu for only three years and have seldom used h.v.l. = 1 or 2 mm. Al for large totals to large areas. We have seen *restitutio ad integrum* as beautiful after a heavy course of h.v.l. = 0.8 mm. Cu as after h.v.l. = 5 mm. Cu. We have produced subcutaneous indurations by sufficient doses of the latter. It takes more roentgens' exposure (measured in air) to do damage with h.v.l. = 5 mm. Cu, but we are not sure whether it takes a larger tissue dose.

(*c*) One wins as good reward for fractionation and protraction with h.v.l. = 1 mm. Al as with harder ray. I am highly enthusiastic about treating small cancers of the face and hands at 400 or 500 r a day instead of with a massive single dose.

(*d*) I will confess to a feeling that h.v.l. = 5 mm. Cu is easier on the patient's skin and constitution for equal effect on the tumor,

[4] Stone's report at this Congress provides a really definite clinical answer as between "deep therapy" and "supervoltage."

but I am still unable to prove that it is not all superstition.

(*e*) Usually the reactions of skin and mucous membrane have seemed slower to come up under h.v.l. = 5 mm. Cu. Yet several cases treated with fractionated h.v.l. = 1 mm. Al have delayed epidermitis a week beyond the expected date.

(*f*) Using h.v.l. = 5 mm. Cu cross-firing the pelvis we are observing reactions in the vagina and uterine cervix that I am sure we never saw when using h.v.l. = 0.8 mm. Cu. Here is the one instance in which I am clinically certain of the superiority of "supervoltage." To accomplish this, we have adopted narrower beams and oblique crossfire that would be, we think, quite ineffective with softer qualities.

CONCLUSIONS

Above the ordinary deep therapy range (h.v.l. = 1 mm. Cu), further increase in hardness produces further increase in percentage depth dose. But the improvement is quantitatively important only for narrow beams or great depths.

The increase in exposure (roentgens measured in the beam) needed to produce an erythema with the shorter wave lengths is no proof of a superior therapeutic effect.

Consideration of the mechanism of absorption of x-ray energy in the body gives little hope of worthwhile change in biologic action on going to wave lengths shorter than 0.05 Å. Very hard cathode rays might give a sharper test than higher voltage x-rays.

Irradiation with fast neutrons may afford a clinical test at the other extreme of pattern of tissue ionization with the same percentage depth dose as supervoltage x-rays.

It seems foolish to split hairs about small differences in quality of x-ray (say between h.v.l. = 1 mm. Cu and 2 mm. Cu), when theoretically and clinically h.v.l. = 1 mm. Cu and 5 mm. or even 10 mm. Cu are so similar.

My clinical impression is that one quality of x-ray is remarkably like another in its effect on the body.

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THE CALIBRATION OF X-RAY DEVELOPER[1]

By G. M. BUSANOVICH, *Philadelphia*

THE high degree of accuracy obtainable with modern x-ray apparatus and methods of exposure requires correspondingly exact work in the dark room to insure uniform results from day to day. However excellent the exposure technic, density and contrast may be impaired or detail may be obscured by fog during development. The chief concern of the processing room is to maintain the extremely fine control of the developing solution that is necessary in producing comparable films of good quality[2] (1).

Development is a chemical process in which the exposed silver halide of the emulsion is reduced to black, finely divided silver by the developing solution. The chemicals are gradually exhausted by the films that are developed, and, to a less extent, by aerial oxidation. The exhaustion of the chemicals must be compensated for by lengthening the development time at definite intervals, that is, after a certain number of films have been developed. The compensation in time must be sufficient to allow for the loss in density and contrast, but must not cause too much fog; for developers, as they are being used, give less density and less contrast, and, to a slighter degree, produce more fog, which is further increased by the longer development.

In the x-ray film, density, contrast, and detail are intimately related and are determined mainly by the exposure technic (voltage, current, length of exposure, size of the focal spot and its distance from the film, efficiency of intensifying screens, etc.). To a less extent they are affected by the developer. With a given exposure, a definite density and a definite degree of contrast are obtainable by development in a particular fresh solution for a definite period. Increase in the length of development of one of two otherwise identical negatives causes an increase in density, and usually in contrast. The chief requirement of the developer is that the contrast and density decided upon as satisfactory, for general purposes, shall be maintained throughout the entire life of the solution. In common practice, this depends upon accurate adjustments of the development time. For a particular purpose, more or less density and contrast may be sought by over- or under-development.

In any competent work with developers, some account is taken of their effect upon density but their effect upon contrast is usually disregarded. Nevertheless this is an important consideration, because as the developer becomes exhausted there is a loss in both density and contrast, but a point is reached at which the latter falls more rapidly than the former. Consequently they should be measured separately.

Fog may be produced during exposure (*e.g.*, by secondary radiation) or in developing. It is axiomatic that during development light must be rigidly excluded from the dark room. Chemical fog must be kept at a minimum by scrupulous care, preventing at all times contamination of the solution by foreign material (soap, metal, dust, etc.). The inevitable increase in development fog as the solution ages is a less serious problem than formerly, owing to improvements in the manufacture of film (2). Nevertheless this factor must be accounted for during calibration of the developer, and this will insure that it has not become an appreciable cause of obscuration.

Only by exact calibration of the x-ray

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[1] From the Research Department of Respiratory Diseases of the Germantown Dispensary and Hospital, Philadelphia; written with the collaboration of Miss Eleanor S. Cooper, research editor.

[2] The calibration of x-ray developer, as described, was made as precise as possible for Dr. F. Maurice McPhedran, in connection with radiographic examination of the lungs, and was applied to general roentgenography under Dr. Thomas P. Loughery.

developer, that is, accurate measurement of its action on exposed film, can all factors be eliminated other than those for which dark-room technic is responsible. This subject has been discussed in various papers (3), but the emphasis has been almost wholly upon density. The Watkins factorial system of development is sometimes recommended, but it depends too much upon the eye of the individual observer, who times the seconds to the first appearance of the image after the film has been placed in the bath. There is no method in general use for obtaining exact figures for ready reference in determining the proper development time. The following method is in part a modification of that described by Weyl and Warren (2), with added emphasis upon contrast and fog, and the construction, from several calibrations, of an average or standard calibration curve. Such a curve will be reliable for reference for several years, or as long as there is no change in the manufacture of the film or developing powder used.

THE CALIBRATION TESTS: PREPARATORY STEPS

The method consists of the exposure of x-ray film under an aluminum stepladder and the subsequent development at regular intervals of strips of the exposed film. The ladder, 14 inches horizontally and 6 inches vertically, consists of aluminum sheets of a thickness beginning at 1 mm. and increasing by 1 mm. for every inch of height, thus making a staircase running from one to six thicknesses. A film, cut 14 inches long by 8 1/2 inches wide, is placed lengthwise in a 14 × 17 inch cassette, two inches below the top of the cassette. Care is taken that there shall be excellent contact between the film and the intensifying screens. The cassette is placed in an upright chest machine (or in whatever type of apparatus is used). The aluminum ladder is supported by strips of adhesive at its corners attaching it to the lead shield (used in making two or more separate exposures on the same film), which has been screwed on the lower half of the door of the plate changer. An exposure is made with the x-ray technic used for a chest of average diameter and average thickness of chest wall (18 cm.—type B).

The film is removed from the cassette in the dark room. As a precaution, because the intensifying screens may not be in perfect condition, one inch is cut off along each side of the film, reducing it to 12 inches (horizontally) by 8 1/2 inches (vertically). The film is then cut into 12 strips each measuring 1 × 8 1/2 inches. Two more 14 × 8 1/2 inch films are similarly exposed, and similar strips are placed in corresponding envelopes marked Exposure II and Exposure III.

Before proceeding further, account is taken of the fact that x-ray film may show objectionable variations in density over its surface if exposure is made at distances of less than 50 inches. Accordingly another 8 1/2 × 14 inch film is exposed under the aluminum ladder, processed, and tested throughout with the densitometer.

The final preparatory step is to develop a "standard strip" from each envelope of the three exposures. In fresh developer, which has stood at least 12 hours after preparation and has not had a single film developed in it except the above, the three strips, marked I, II, and III, are developed for the period of time selected (5 or 6 minutes) at a temperature of 65 degrees F. (Temperature measurement must be accurate within one-half degree.) Only in this way, by having the strips of the three x-ray exposures entirely separated, and by having a standard strip developed for each, can the possibility of error in exposure technic be eliminated. The method is also a check upon possible variability in the speed of the film, which may be appreciable in films of the same emulsion number, and should not exceed 5 or 6 per cent.

At the bottom of each strip there is a two-inch clear space of film, which was protected during exposure by the lead shield on the door of the plate changer. This is used for the separate measurement, in the standard strips and in the test

strips as they are developed, of the generally diffused density caused by fog.

THE CALIBRATION TESTS

With tanks of 10 gallons' capacity or larger, the test strips are developed at intervals spaced by the development of 50 large films or their equivalent. (For practical purposes the following figures in terms of 14 × 17 inch film are used: one 8 × 10 inch film equals 2/6, one 10 × 12 inch film equals 3/6, and one 11 × 14 inch film equals 4/6 of a 14 × 17 inch film.)

All exposed strips are developed vertically in the tank with the lightest part (the least dense step) uppermost. The solution is agitated previous to, and for the first minute of, immersion. The periods for rinsing, fixing, washing, and drying are kept constant as for the usual run of work.

Density and Contrast.—The equivalent of 50 large films having been developed, three strips are taken from the envelope of Exposure I and marked with lead pencil I-1, I-2, and I-3. They are developed for 6, 6 1/4, and 6 1/2 minutes, respectively, at 65 degrees F. (if the standard strips were developed for 6 minutes). After drying, they are compared with standard strip I. With a densitometer such as that developed by Weyl and Warren (2), readings are obtained of the densities of all six steps in the standard strip and in the three test strips. The results are tabulated, and the density readings of the corresponding steps of the test strips and of the standard are compared. The development time of that strip in which the densities in steps 2, 3, 4, and 5 approximate most closely to the corresponding steps in the standard, is the correct period to compensate for exhaustion of the capacity of the developer to bring out density in an exposed film. The difference between the density readings of steps 1 and 6 will give a figure that has been found to be entirely satisfactory as used in calibration for the approximate contrast.

For example, the densities shown in Table I were obtained from one series of strips.

TABLE I

	Standard I 6 minutes	I-1 6	I-2 6 1/4	I-3 6 1/2
Step 1	2.24	2.02	2.14	2.22
Step 2	1.50	1.28	1.37	1.48
Step 3	0.92	0.71	0.79	0.88
Step 4	0.60	0.46	0.50	0.58
Step 5	0.36	0.27	0.30	0.34
Step 6	0.24	0.20	0.20	0.23

As can be seen at a glance, I-3 compares most favorably with the standard. Subtracting step 6 from step 1, to arrive at the approximate contrast, we have 2 for the standard, 1.82 for I-1 (same development time as for the standard), 1.94 for I-2 (one-quarter minute more), and 1.99 for I-3 (one-half minute longer than the standard). Again, I-3 is closest to agreement with the standard strip. The next 50 films are accordingly developed for 6 1/2 minutes.

Usually, in the early tests, the strips will compare very closely in all the steps, but as more films are processed variations in the density of some steps will occur. The developer must be discarded when any one of the steps 2, 3, 4, or 5 varies by 10 per cent or more from the corresponding step of the standard, even though all the others agree exactly. Beyond this, the density difference will be appreciable (1, 2) in serial films.

Also when the contrast, determined as described, varies by 5 per cent or more, the solution should, as a general rule, be discarded. This is perhaps the only entirely safe guide, but, on the other hand, as contrast falls off more rapidly than density, it may be too expensive or otherwise inconvenient to replace the developer when loss in contrast begins to be excessive. In such cases, the technician may undertake to effect a compromise between density and contrast.

For example, during a calibration of the developer at the Germantown Hospital, a series of figures was obtained showing satisfactory similarity in density and contrast up to the number of 200 large (14 × 17 inch) films, when an unwanted loss of

contrast was noted. According to density (a density reading of the intermediate steps on the test strips), the development time should have been 7 minutes, but the variation in contrast was too great for good comparability. As regards contrast, the development time should have been 7 1/2 minutes, but this gave too much density in the intermediate tones. A development time of 7 1/4 minutes was found to be a satisfactory compromise, and was correspondingly effected in all subsequent readings, until at about 450 films the variation in contrast had become too great. At that point the developer was discarded and fresh solution was prepared.

Fog.—Most films that are satisfactory for density and contrast are likely to be free of objectionable fog. Nevertheless it is clear that development fog will contribute most to obscuration in the lighter (less dense) fields, and may be missed if only the darker areas are tested for density. This factor must be determined separately and ruled out in calibration by exact measurement, which may also be useful if any question of obscured detail should arise.

Accordingly density readings are made of the two-inch clear (unexposed) film at the lower end of the test strips and compared with a similar reading on the standard. With film now available, fog should not measure more than 0.15 after development in fresh solution for 8 or 9 minutes (2). For this reason, a development time of 6 minutes is commonly chosen. As the calibration tests proceed, the increase in fog caused by the exhaustion of the developer and the lengthening of development time (but offset somewhat by the routine replenishment of the solution, described below) should not exceed a density reading of 0.10. This is rarely observed before several hundred films have been developed, but it may occur even before loss of contrast has become conspicuous.

Fog may also be caused during development, as mentioned above, by light leaks, or by contamination of the solution.

REPLENISHMENT OF THE SOLUTION

Thus for every group of at least 50 large (14 × 17 inch) films processed, three test strips are developed for different periods, ranging from the interpolated development time of the preceding strips to one-fourth and one-half minute longer, and are compared with the corresponding standard strip. Meanwhile the developer in the tank must be maintained at a constant level by the addition of fresh developer. *The fresh solution must be added in small quantities at regular intervals.* As each 14 × 17 inch film absorbs approximately 1 1/2 ounces of developing solution, it is excellent practice to add one pint of developer after every 10 large films or their equivalent developed. If this is insufficient to maintain the constant level of the solution, more than one pint should be added at the same intervals, until by variation the correct amount that is necessary to maintain the required level is found. To avoid possible dilution, the capacity of the tank should be determined by actual measurement; one tank, which had been rated by the manufacturer at 15 gallons, was found to hold 17 1/2 gallons.

The calibration tests are continued until the variation in contrast or the increase in fog becomes too great, when the solution is discarded. Usually, with a 15- or 20-gallon tank, no great variation in contrast occurs until the developer has processed the equivalent of several hundred large (14 × 17 inch) films. When a considerable variation in contrast is found in developer that is relatively fresh, the results should be checked and re-checked, but this is unlikely to occur unless the solution has been replenished by adding, at irregular intervals, irregular amounts of fresh developer.

THE CALIBRATION CURVE

As each test is carried out, the number of films or total film area developed in the solution and the development time of the strip that most closely agreed with the corresponding standard strip are recorded. With these data from all the tests, points

are plotted on graph paper, with the periods of development as the ordinates and the number of films processed as the abscissæ. The more points are obtained, corresponding to the number of tests made, the more accurate will the graph be. Through the points a smooth curve is drawn; this is the calibration curve showing the rate of exhaustion in the developing solution that has just been used.

Each calibration should be completed within one month to avoid deterioration of the developing solution by aerial oxidation and, possibly, changes in the exposed films. One calibration is not sufficient to use for exact prediction of the proper time of development at any given interval. At least three must be taken under the same conditions, and the average of the three may be considered a standard calibration curve. Theoretically, all three curves should coincide, but irregular oxidation of the developer, changes in chemicals of the developing powders, and other variable factors cause some deviation from coincidence (Fig. 1).

The average or standard calibration curve (Fig. 2) gives information for development technic that may be said to correspond to the more complex calculations for accurate exposure technics that have been made in terms of current, voltage, the physical properties of the chest, the type of x-ray apparatus, etc. As these were charted for the x-ray technician, it was thought that the calibration curve of the x-ray developer might be similarly useful in dark-room procedure. In actual practice, it has been found more exact and easier to refer to than the charts in common use.

The calibration curve may accordingly be drawn on graph paper and hung in the dark room. The technician then has only to keep an accurate count of the films developed, while the correct development time is found, whenever desired, by tracing a vertical line from the number of films until it meets the curve, and from that point tracing a horizontal line until it meets the ordinate, where the correct time of development is found. This must be

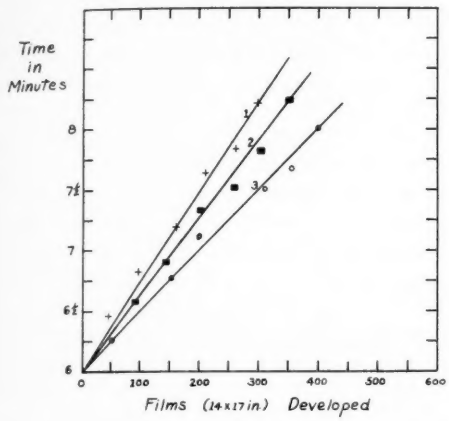

Fig. 1.

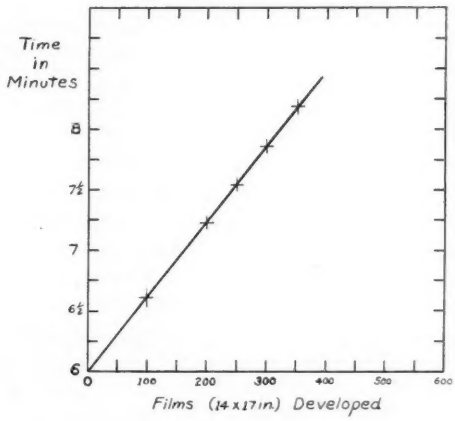

Fig. 2.

Fig. 1. Three calibration curves plotted on one graph. The curves were obtained from three batches of one brand of developer mixed in a 17 1/2-gallon tank. It is from these data that the average or standard calibration curve is obtained.

Fig. 2. The standard calibration curve, obtained as follows: The three time-intervals of Curves 1, 2, and 3 are found on the 350-films ordinate in Figure 1. These (8 1/2, 8 1/4, 7 3/4 minutes) are added and the result is divided by three. This point (8.2) is found on the corresponding ordinate in Figure 2. Similarly, "average" points are found on the 300-, 250-, 200-, and 100-films ordinates. Through these is drawn a straight line, which becomes the average or standard calibration curve.

interpolated to the nearest quarter-minute (at 65 degrees F. ± 1/2 degree).

The only remaining difficulty, if undertaken at all, is the compensation necessary for the irregular loss of contrast as the developer ages. This problem can be avoided by discarding the developer as soon as an appreciable loss of contrast appears, but this step may be unduly expensive, or, in the midst of work, impossible. Perhaps an exact and practical method to eliminate the uneven loss of contrast might be found in the use of the same developer for replenishment until just before loss of contrast may be expected from experience, and, from that point, the addition of developer of higher contrast as required. This method appears worthy of consideration and trial. Nevertheless, there is little to object to in the method of compromise, except that much depends upon the ability of the technician to effect a satisfactory relation between density and contrast.

APPLICATION

Calibration may be adapted to other methods of development. One that has recently come into use consists of regular replenishment of the solution with a stronger, modified developer, while time and temperature remain constant (4). It is possible that the best results will finally be obtained by a combination of methods, that is, by adjusting the time in the usual way at first, then later replenishing the solution with the modified developer while continuing to increase the development time, but at longer intervals. In any case, calibration will serve as an accurate check on the exact effect of the development upon the radiographic image.

The calibration of developer has been used in general roentgenography, and particularly as an aid to the accurate work that is essential to x-ray examination of the chest (5). It is applicable to other kinds of photography (6). For the most precise results in developing, the solution should be tested daily by the method described. However, in the calibration curve obtained as described the error is insignificant. Once plotted, it may be relied upon for one or several years as an accurate and convenient device for determining, at any interval, the length of development time that will bring out the full range of density, contrast, and detail in the exposed film.

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JUVENILE OSTEOCHONDROSIS[1]

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SINCE 1903, when Osgood first described the roentgenologic appearance of a non-infectious, non-traumatic rarefying lesion of the tibial tubercle, there has arisen extensive and confusing literature on similar conditions in other growth centers. To-day, there is hardly a bone, except possibly those of the head, that has not been described as the site of this lesion. Such lesions were considered by various authors in earlier days as epiphysitis, chronic, non-suppurative, non-tuberculous, non-traumatic, aseptic, etc., and the original author honored by ascribing his name to the lesion. Thus, the same lesion may be called *Legg's disease* by one, *Perthes' disease* by another, and *Calvé's disease* by still another. For the same reason, we have *Calvé's disease of the primary centers of ossification* and *Scheuermann's disease of the secondary vertebral centers.* It is little wonder then that the average medical man is overwhelmed by this nomenclature and constantly in need of an interpreter when referring to this subject. Struck by the similarity of these various conditions under different names, authors began to classify them under general heads. Harbin, in 1930, and Brailsford, in 1935, with certain differences grouped them under the head of *osteochondritis*. Harbin more recently proposed the term *osteochondrosis*, pending a more satisfactory classification. Rigler, in his "Outline of Roentgen Diagnosis," also included most of these lesions under the head of *osteochondrosis*. The name *juvenile osteochondrosis* is more specific, since it excludes those definitely post-traumatic malacias, such as Kümmel's, Preiser's, and Kienböck's disease, which do occur in adult life. Osteochondrosis is a disease of the growth centers, and has its inception and activity before growth ceases.

The etiology still remains in doubt, though many theories are advanced. The theory that it is of endocrine origin has many advocates. Schaefer, *et al.* examined 258 endocrinopathic cases and concluded that *juvenile chondro-epiphysitis*, a term they prefer, is pathognomonic of hypothyroidism. Brailsford is satisfied that trauma is a definite factor but that some other unknown element is essential. Hereditary infections and endocrine disturbance undoubtedly play some part, but, clinically and experimentally, the etiology is unsettled. King proposes that it is not a distinct entity, rather a syndrome of clinical signs and symptoms, with a characteristic roentgenologic picture, and that it is due to a number of different causes. The pathology, too, is unsettled. The fact that the disease lasts over a long period, during which time it is seen by different observers at various stages, probably accounts for this situation.

The signs and symptoms vary. The acute stage occurs when that particular growth center involved is most active, *i.e.*, when the cartilage is undergoing ossification. The primary centers are affected in the first decade, the secondary centers in the second decade. They all last an indefinite length of time and terminate at fusion or cessation of growth. The symptoms are usually very mild; most cases pass unnoticed. Fever, if present, is so mild that it is often not noticed. There is usually a history of trauma, which aggravates the condition and often calls attention to it. In some cases, a deformity results which may be very definite, such as occurs in spinal lesions, or this deformity may be so well compensated that it passes unnoticed on ordinary examination.

As military surgeons we seldom, if ever, see the acute cases in soldiers. However,

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this stage is seen in the children of military personnel and in CCC boys. The deformity, however, particularly that of the spine, the hip, or the knee, can readily be overlooked in the applicant for enlistment or work and become evident in adult life as a disability. Often the applicant himself is not aware of his condition, or the deformity is so slight that it passes unnoticed. However, when subjected to the stress and strain of military life or other hard work, a disability may result. After a long hike or indulgence in athletic sports, the individual may begin to complain of pain in the legs or back. Perhaps, after some slight injury, he is sent to the X-ray Department and the condition is revealed.

Interested by the frequency with which this condition was found, particularly in the spines of applicants for enlistment and appointment in the Army, and impressed by the fact that the average young medical officer is not familiar with its military significance and the terminology, a study of the pathologic file for the past four years in the X-ray Department, Station Hospital, Fort Benning, Georgia, was made, for the purpose of presenting the findings at a Staff meeting. The following cases were selected and reviewed:

Osteochondrosis of spine, Scheuermann's disease	23
Osteochondrosis of spine, post-traumatic, Kümmel's disease	3
Osteochondrosis of femoral head, Legg-Perthes-Calvé's disease	3
Osteochondrosis of tibial tubercle, Osgood-Schlatter's disease	3
Osteochondrosis of tarsal scaphoid, Köhler's disease	1
Osteochondrosis of epiphysis of tarsal scaphoid	1
Osteochondrosis of os calcis, Sever's disease	3
Osteochondrosis of head of second metatarsal, Freiberg's infraction	1
Osteochondrosis of internal condyle femur, osteochondritis dissecans	2
Osteochondrosis of carpal scaphoid, Preiser's disease	6
Osteochondrosis of semilunar, Kienböck's disease	2

One case of spondylolisthesis is mentioned because this condition is thought by some to be due to osteochondrosis. This patient was an applicant for enlistment who had the typical conformation and gait.

Osteochondrosis of the Vertebræ.—Described in 1921 by Scheuermann, it is known as *Scheuermann's disease, juvenile* or *adolescent kyphosis, vertebral epiphysitis, schoolchild kyphosis,* etc. Of the 23 cases so diagnosed, six were CCC boys, five were dependents of military personnel, six were applicants for enlistment, three were recruits, two were applicants for commission, and one was an applicant for West Point. This condition usually develops between the ages of 15 and 17 years and terminates when fusion takes place. The vertebræ usually affected are those between the seventh dorsal and third lumbar, which vertebræ because of static demands are subjected to the most pressure. Some early cases show only slight irregularity of the epiphyseal plates, particularly on the anterior superior, and inferior margins. This irregularity may increase until there is an appearance of fragmentation with cup-shaped depressions on the superior and inferior surfaces of the bodies. In more advanced cases, the bodies become wedge-shaped. This wedging process causes a deformity which is permanent and may vary from a slight exaggeration of the normal posterior curvature to a cone-shaped rigid kyphosis. During the acute state, there is atrophy or demineralization. This, with the wedge shape of the bodies, may arouse a suspicion of Pott's disease. In order to maintain a correct posture, a patient may force the cervical, upper dorsal, and lumbar vertebræ into a position of lordosis. This was most marked in the applicant for West Point. One case, a CCC enrollee, roentgenographed after a slight injury, was thought to have a fracture (Fig. 1, Case 1) of the anterior, superior margin of the body of the second lumbar vertebra. However, closer attention to the whole spine (Fig. 2, Case 1) revealed a definite kyphosis with irregularity of the epiphyseal plates and typical wedge-shape of the twelfth dorsal vertebra.

Symptoms during the active stage may be mild or entirely absent. A painful weariness which increases on work is described by Scheuermann. In only a small proportion of our cases was there a history of painful back. A definite history of trauma was obtained in only one case; the rest did not remember or concealed a history of previous injury. The majority of our cases were roentgenographed because of poor posture or deformity. Most of them were well developed and muscular. One boy of 17, the son of an officer, had the typical round back and frequently complained of pain, but he played an excellent game of golf. The three post-traumatic cases (Kümmel's disease) had the same roentgenologic appearance as the adolescent type. However, they all occurred in older soldiers and had definite histories of injury; two had had slight compression fractures. Two patients showed the characteristic Schmorl's nodes (Fig. 3), one of whom had cord symptoms very suggestive of pressure due to herniation of the nucleus pulposus. Needless to say, any applicant for enlistment or for commission presenting any of these defects is rejected.

Osteochondrosis of the Femoral Head.—Described by Legg, Perthes, and Calvé in 1910, and known as *Legg's disease*, *Perthes' disease*, and *Calvé's disease*, *osteochondritis*

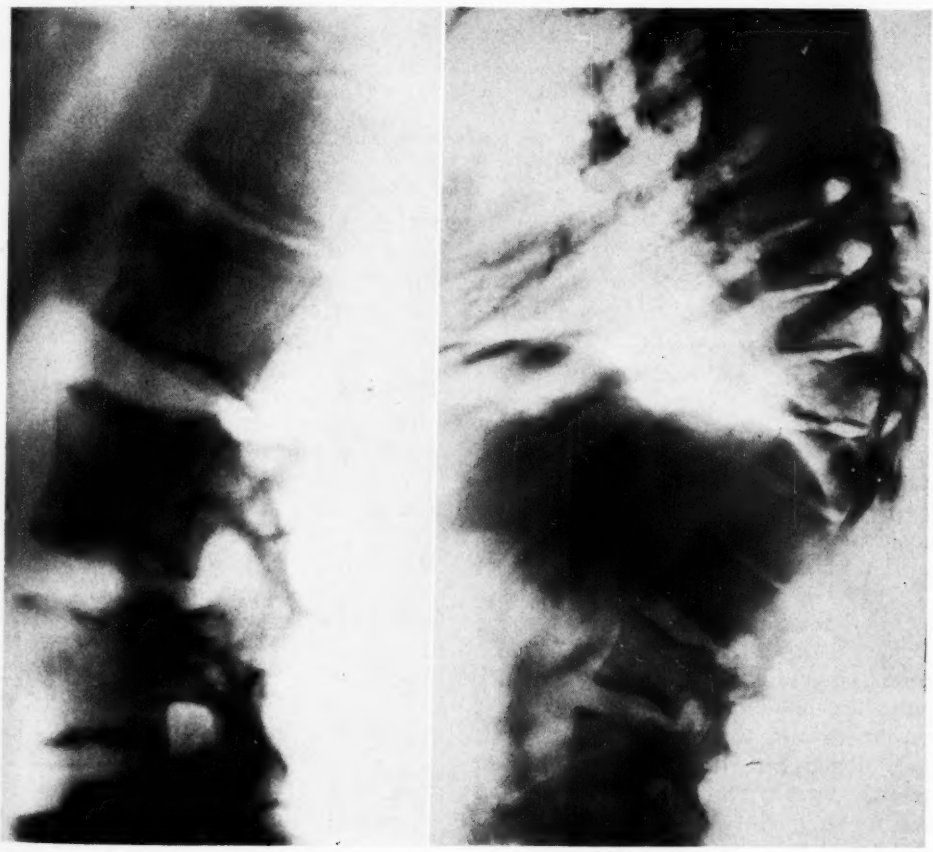

Fig. 1. Fig. 2.

Figs. 1 and 2. Case 1. Osteochondrosis of vertebræ.

Fig. 3. Fig. 4.

Fig. 3. Osteochondrosis of vertebræ, post-traumatic, Schmorl's nodes.
Fig. 4. Osteochondrosis of femoral head.

coxæ juvenilis and *osteochondritis deformans juvenilis*.

Harbin states that this is probably more frequently seen than any other type of osteochondrosis. This was not borne out in our investigation, since only three cases were found in the past four years, one a soldier, one a CCC youth, and one a civilian employee. The soldier was 21 years of age and was admitted into the hospital because of an injury to his left knee. The ward surgeon noted a limp and a tilted pelvis and called for a roentgenologic examination of the knee and pelvis. The left knee was negative, but the typical deformity of the left femoral head was noted (Fig. 4). The patient gave no history of previous injury to the hip. On inquiry at his company, it was ascertained that, before injuring his knee, he marched, drilled, and indulged in athletic sports without sign of a limp and with no complaint. The other, a powerfully built athletic CCC enrollee, claimed he hurt his hip and was sent to the hospital for diagnosis. A well healed bilateral osteochondrosis was seen. The third case was a man 60 years of age who had done hard labor most of his life. On examination for a civil service position, a slight limp was noted. He, too, had a bilateral osteochondrosis which did not interfere with his livelihood.

The active stage of this condition is usually seen between the ages of three and 15 years, possibly later, and is rarely seen at Army posts. During this stage, the only complaint may be a slight limp; however, there may be pain and some tenderness. On physical examination, there will be found limitation of adduction and rotation. In the early stage, the x-ray examination may be negative: later the characteristic appearance is noted—rarefaction of the head and area near the epiphyseal line. Still later the head becomes flattened and possibly fragmented, the epiphyseal line becomes dense and more horizontal, and the neck becomes thicker and shorter. The deformity may be a very large circular head or have a mushroom appearance with

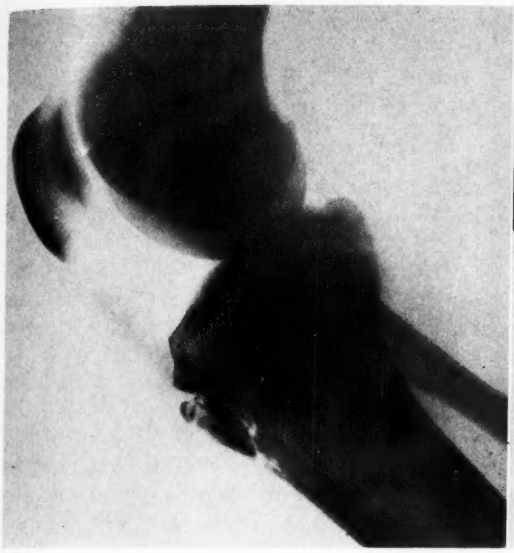

Fig. 5. Fig. 6.

Fig. 5. Osteochondrosis of tibial tubercle.
Fig. 6. Osteochondrosis of internal condyle of femur.

fragmentation, with small fragments which appear to be crowded out of the acetabular rim. The articular space is either normal or widened. It must be differentiated from tuberculosis of the hip, coxa vara, congenital deformities, and slipped epiphyses. During the active stage, the differentiation from tuberculosis may be difficult; however, the clinical course of tuberculosis with its sharp pain, marked limitation of motion, fever, and muscular wasting is much more severe. If tuberculous, erosion is present, the joint space is decreased, and rarefaction is more pronounced. From the others, the characteristic appearance of the film suffices.

That these soldiers with deformities of the femoral head pass the physical examination on entrance into the Army is not a reflection on the examiner. Many cases compensate so well that the limp is not noted. Even the waddling gait in bilateral cases may fool the examiner. One case in particular is recalled. A young officer passed through the training camp, went overseas with a combat division, and was cited several times for bravery in action. Returning to the United States, he got along very well until 1922 when he was sent to the Army and Navy General Hospital, Hot Springs, Arkansas, for ar-

Fig. 7. Epiphysiolysis. (Compare with Figure 4.)

Fig. 8. Osteochondrosis, head of second metatarsal bone.

thritis. A bilateral osteochondrosis of the femoral head was found.

Osteochondrosis of Tibial Tubercle.—First described by Osgood in 1903 and by Schlatter in 1908, this condition is known as *Osgood-Schlatter's disease* or *tibial tubercle epiphysitis*. This usually affects well developed boys at puberty, the average age about 13 years. Consequently, it is seen by military surgeons only in children of the Army personnel. Of our three cases, two were boys and one was a girl; all healthy, strong, athletic children with no definite history of direct trauma and no signs of endocrinopathy. Cole, in a study of 40 cases, found that 19 gave no history of direct trauma, 11 gave a history of direct trauma, and eight stated that indirect trauma such as running and jumping antedated the symptoms. The usual symptoms are pain over the tibial tubercle, increased on running and walking. On physical examination, tenderness on pressure and some swelling are noted over the tibial tubercle. The condition is usually bilateral. Roentgenograms may show nothing, but there is usually some enlargement, some irregularity, or fragmentation of the tubercle. The symptoms clear up in time, but they may persist from three to five years. One case, which we were able to follow, was seen in the acute stage in 1935; films made three years later showed a complete return to normal (Fig. 5). This case was immobilized for six weeks: no other treatment was given.

Osteochondrosis of the Internal Condyle of Femur.—Described by König in 1888, it and *Kümmel's disease of the spine* are probably the only conditions of this kind recognized before the discovery of the x-ray. Known as *osteochondritis dissecans*, it is always found in the internal condyle of the femur, but Miller has reported a similar condition in the capitellum of the humerus. It is mostly seen in males between the ages of 16 and 21 years and there is usually a history of trauma. Köhler thinks that the most probable cause is injury to the popliteal artery by many small, often repeated wrenches. Some patients give a history of discomfort in the knee off and on for several years, others may complain of sharp pain and locking of the joint after some injury. The roentgenographic appearance is characteristic; an oval-shaped shadow denser than the surrounding tissue surrounded by an area of translucency on the internal condyle. These small dense bodies give the impression that they could be picked out easily. Eventually, they may become loose bodies in the joint and give rise to the characteristic signs of joint mice.

Two cases came under our observation. One, a soldier, aged 20, who injured his left knee in May, 1937. The typical picture of osteochondritis dissecans was found on the film. He remained in the hospital until the acute symptoms subsided, when he returned to duty: he has not been seen since. The other, a CCC boy, aged 19, was admitted to the hospital with the history of having struck his right knee with the blunt end of an axe about three weeks prior to admission. When first seen he had a decided limp and complained of a dull aching pain on walking. On examination, there was some tenderness over the internal condyle (Fig. 6). Roentgenograms showed the typical appearance of osteochondritis dissecans. A few weeks later, when the

pain in the knee had subsided, he complained of pain in the right hip. On films, a definite coxa vara was found. This was then considered a Legg's disease (Fig. 7). However, the head appeared to be normal, while the neck appeared to be displaced upward along the epiphyseal line. A diagnosis of epiphysiolysis, or slipped epiphysis, was later made. The boy denied any previous injury to the knee and did not remember any former injury to the hip. When the epiphysis slipped is only a conjecture.

Osteochondrosis of Os Calcis.—Described by Sever in 1912, and known as *apophysitis, epiphysitis,* and *Sever's disease,* this is usually seen in boys between the ages of 8 and 14 years. Only two cases were seen in the past three years at this post, both active, healthy boys, the children of officers. The chief complaint was pain in walking. On examination, some tenderness of the heel was noted on pressure. Flexing the feet caused pain by stretching the tendo achilles. Roentgenograms may show very little, but they may show enlargement, irregularity, and fragmentation of the epiphyseal line with areas of rarefaction and condensation adjacent.

Osteochondrosis of the Head of the Second Metatarsal Bone.—Known as *Freiberg's infraction, second Köhler's disease,* or *juvenile deforming metatarsophalangeal osteochondritis,* it was first described by Freiberg in 1914. Only one case has been seen at this hospital, although a similar case was seen at another station but not reported. Lewin states that there are only 65 cases reported. Our case was a soldier, aged 21, who complained of pain in his foot when he marched or drilled, and gave a history of an injury four years before. On the film, the characteristic flattening of the metatarsal head was noted, with some thickening of the neck, fragmentation of the epiphysis, irregularity in the epiphyseal line, and widening of the joint space (Fig. 8). This soldier was discharged on disability.

Osteochondrosis of the Tarsal Scaphoid.—Described by Köhler in 1912, it became known as *Köhler's disease* and *tarsal scaphoiditis.* It is usually seen in children from three to ten years of age, the common complaint being pain and swelling over the scaphoid bone. The roentgenogram is all-important in the diagnosis, namely, loss of bone structure and marked increase in density. According to Köhler, the bone returns to normal without treatment in one or two years. Our case (Fig. 9), a soldier, age 20, still shows marked condensation and sclerosis. He has, however, other signs of interrupted bone growth, such as multiple parallel striations in the metaphyses of most long bones and concentric ring shadows in the irregular bones and vertebræ. He also gave a history of injury to the foot as a child. King points out the rather frequent occurrence of striations in the metaphyses with osteochondrosis, particularly that of the tarsal scaphoid.

Fig. 9. Osteochondrosis, tarsal scaphoid. Note: Sclerosis of trochlea of talus, articular surface of tibia, epiphysis of calcaneus, ring shadows in cuboid, and cross-striations in metatarsals and phalanges.

The other case, a girl aged 15, complained of painful feet on exercise. On examination the feet were found to be flat and slightly everted. The scaphoid bones were prominent and definitely tender on palpation. On roentgenograms, the tuberosities of the scaphoids protruded and

the accessory os tibiale externum was present bilaterally. The bone at the junction of the accessory scaphoid presented

Fig. 10. Osteochondrosis of epiphysis of tarsal scaphoid.

the moth-eaten appearance of localized demineralization characteristic of osteochondrosis of the carpal scaphoid (Fig. 10).

This condition is relatively rare. Haglund first described a similar lesion in 1908. King looks upon the accessory scaphoid as an epiphysis of the scaphoid and calls this condition *osteochondritis of the epiphysis of the tarsal scaphoid*. In this way it differs from Köhler's disease which affects the primary center of ossification and hence occurs earlier in life.

Osteochondrosis of the Carpal Scaphoid.—First described by Preiser in 1910, this is known as *Preiser's disease, post-traumatic osteitis, post-traumatic malacia*, or *post-traumatic osteoporosis*. It is usually found in adults with a definite history of trauma, usually a fracture: all cases here considered followed fractures. All had the same characteristic symptoms—pain on use and definite tenderness over the scaphoid bone. The roentgen appearance depends on the time the condition is seen. All our cases showed small circular areas of osteoporosis around old ununited fractures (Fig. 11).

Osteochondrosis of Semilunar Bone.—This was first described by Kienböck in 1910, therefore, it is known as *Kienböck's disease, post-traumatic osteitis, post-traumatic malacia*, or *post-traumatic osteoporosis*. The symptoms are pain and swelling in the wrist with tenderness over the semilunar bone. Most commonly seen in laborers with a history of long-continued indirect trauma to the hand, as by use of an air drill or automatic riveter. On the film are seen small circular areas of osteoporosis in the semilunar bone. Two such cases, both in soldiers, were seen in the past two years.

While this paper does not add anything in particular to the study of the scientific aspect of the condition, it did bring out the rather startling fact that more than half of the cases of juvenile osteochondrosis seen at this Army Hospital affected the spine. This may be explained by the fact that all patients examined, all applicants for enlistment, for commission and for West Point, who show any postural deformity are sent to the X-ray Clinic for examination. The same results would obtain, no doubt, if all defects in posture seen in high schools or colleges were surveyed roentgenologically.

The recognition of the deformity that results from an old osteochondrosis of the spine usually presents no great difficulty. However, in the early stages and later, when fragmentation of the epiphyseal plates is marked, the diagnosis may become quite difficult, particularly after a history of injury to the spine.

Excluding the spinal cases and the one so-called osteochondrosis of the epiphyses of the tarsal scaphoids, and the lesion of the

head of second metatarsal bone, the other cases were the average run seen in any x-ray clinic. The unusual occurrence of

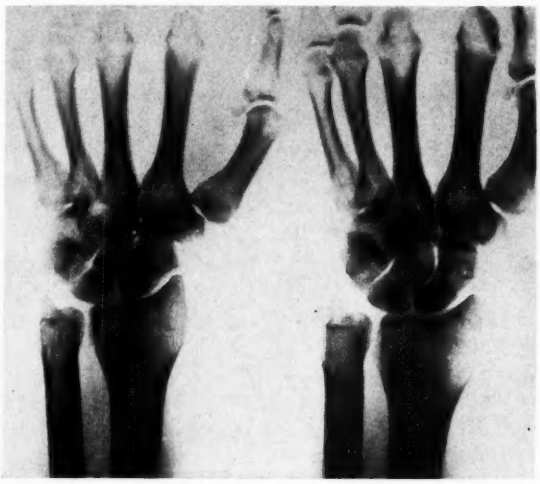

Fig. 11. Osteochondrosis, carpal scaphoid, post-traumatic.

osteochondritis dissecans in the right knee and epiphysiolysis of the right hip, and the association of tarsal osteochondrosis with multiple ring shadows and cross-striations may be more than coincidental.

This study further emphasizes the wisdom of some large industrial firms in making roentgen surveys of the spines of all workmen they hire. These films showing the condition of the spine before an injury, actual or alleged, may be just as important to the employee as to the employer. To be able to establish definitely that this anomaly and that defect was, or was not, present before an injury may mean the saving of much money by an employer, or doing an injustice to an employee.

The fact that most doctors are thoroughly in the dark concerning this condition has long been evident. The reason is obvious; no other condition, not even arthritis, presents such a confusing babel of terms. Until we no longer hear of *Calvé's* and *Scheuermann's disease of the vertebræ* or *Preiser's* and *Köhler's disease of the carpal and tarsal navicular*, we can hardly expect the average medical man to follow us, let alone understand us. The orthopedist, the roentgenologist, and the medical student must speak the same language. We must continue to honor the pioneers who first recognized these condi-

Fig. 12. March foot. Compare with Figure 8.

tions, in the history of roentgenology, but not in the terminology.

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DISCUSSION

James J. Clark, M.D. (Atlanta, Ga.): There is very little I can add to Dr. Stammel's discussion. I have had the opportunity of reading his paper, and he made an excellent review of the literature. I think his point concerning the confusion of terms, the numberless names that are in the books to cover this disease and that disease, is well taken. I believe he is right when he states that it should all be classified under *osteochondrosis* and that is far enough to go.

He is also correct that the roentgenologist should recognize these various forms of osteochondrosis. I know that in the courts every year I see a good many cases that are going on to damage suits because somebody called something a fracture or called a hip joint an injury when it is nothing but a condition that a man has had ever since childhood.

Personally, I agree with the theory that this is an endocrinologic disease. I cannot prove it, but I have felt for a long time that it must be some glandular disorder that affects the growth of the epiphyses during the growing period of a young person's life.

I have one thing more to say. Dr. Stammel is spine-conscious, and I think knowing him and knowing his work has made me a little more spine-conscious, also.

M. J. Hubeny, M.D. (Chicago): The particular case of interest is the one showing the parallel sclerotic changes in both ilia, situated below the crests, which were tentatively considered as "marble bone disease."

We have three such cases in one family in which we have additional findings in some of the long bones comparable to lines usually construed as growth lines, indicating periods of probable systemic diseases and, in addition, somewhat comparable findings in the bodies of the vertebræ. We found on inquiry that these children received phosphorized cod liver oil over a period of several years during certain seasonal periods and since it is a well known fact that the heavy metals might be deposited in the long bones, it occurred to us that the lines aforementioned might be due to the phosphorized oil.

In order to be more certain of this theory we wanted to examine the parents to exclude definitely the possibility of familial trends; however, we were unable to get the co-operation of the parents. We hope that at some future time we shall be able to prevail upon them to submit to desirable x-ray examinations.

I wonder if Dr. Stammel could inquire into the history of the case he presented to find out whether phosphorized cod liver oil had been taken, although it is my under-

standing that this particular patient is an adult.

At this time I would like to make mention of three cases in which we had findings comparable to osteochondritis dissecans involving the upper surface of the astragalus near the internal malleolus. Again, it is hoped to re-contact these cases for verification.

J. H. KITE, M.D. (Atlanta, Ga.): Col. Stammel has shown us some interesting end-results in young adults. I have followed a number of children with this condition by serial x-rays for ten or more years. They occur more frequently in boys than girls, the ratio being about 3 to 1. Most cases begin between four and eight.

This condition was first described independently in 1910 by Legg, Perthes, and Calvé. To avoid the use of proper names it has since been called *osteochondritis deformans juvenilis coxæ*. It is also referred to as *coxa plana*, but I prefer the descriptive term, *flat-headed femur*.

Many theories have been advanced as to the cause of this condition. Clinically, trauma does seem to play a part. Nearly every patient recalls some trauma, usually of a minor nature, which he thinks is responsible for the onset of the condition. We never see the condition following a trauma severe enough to fracture the hip. The minor trauma may injure the nerves or blood vessels and cause certain changes to take place, probably in the circulation of the hip. It seems that there is a loss of circulation to the epiphysis for the head of the femur. The head becomes more dense, which may be due to the fact that it may attract more calcium to itself, or this change may be relative. There may be an increased circulation in the neck of the femur causing a rarefaction of the bone in this region. I do not believe that this condition is the result of a congenital abnormality, as some claim, because I have x-rays of early cases in which practically no change can be seen in the contour of the head. As the condition progresses the dense head becomes fragmented and the bone salts are removed from along the edges of the epiphysis. This leaves a smaller mass in the center with a space about it containing almost no bone salts, while along the edges bone salts are again being deposited. This process continues until all of the sequestrum is removed and only a radiolucent area is left in the center. New bone is laid down over this area along the cartilage on the superior surface, restoring a definite outline to the head, and finally after about two or three years the head is restored to about normal density, if weight-bearing has been prevented. During this same time changes take place in the neck of the femur. There is some absorption of bone salts from the neck, and the neck becomes shorter and broader, probably due to compression from weight-bearing.

It is thought by some that this change is due to some endocrine disturbance, and some of the patients do suggest this, and we know that certain endocrine conditions do show changes in the epiphysis, but these findings are not frequent enough and typical enough to be accepted as the common cause of "flat-headed femur."

Occasionally pyogenic infection of the hip will give an appearance somewhat similar, but this is not the routine cause. Infection of a very low grade in the surrounding soft parts may be a factor in some cases.

The treatment is to avoid weight-bearing during the plastic stage when the bone is soft, and to avoid weight-bearing until the x-ray examination shows almost normal condensation of bone. I do not believe it is necessary to immobilize these patients in plaster. They can avoid weight-bearing by staying in bed, but this is difficult to carry out at home, and the condition does not warrant the expense to an institution to keep them in bed. The next best treatment is to avoid weight-bearing by using a walking caliper brace, with a cork lift under the opposite shoe. The patient sits on top of a ring at the upper end of the caliper. The foot swings free and cannot touch the ground. When patients cannot afford a brace, the third method is to have

a cork lift under the shoe of the normal foot and let the patient walk on crutches, without bearing weight on the affected side. This is the method I commonly use, and they usually obtain a hip free from pain and with free motion in all directions except for a very slight limitation in full abduction and slight limitation in full inward rotation. The x-ray examination still shows some flattening of the head, but a smooth and almost normal contour to the head.

Lieut. Col. CHARLES A. STAMMEL, M.C., U. S. Army (*closing*): Regarding the treatment, there is not much to be said. Most cases get better, or the symptoms subside at cessation of growth. In the spine, however, the deformity may be prevented if hyperextension is applied during the stage of activity. This condition is primarily a condition of adolescence. What I have been interested in, from a military standpoint, are the deformities that result.

Regarding the literature, one writer, King, writing of Perthes' disease alone, had 436 references. This gives one an idea as to how much has been written on the subject.

In regard to that case of osteochondrosis of the tarsal scaphoid which in addition, showed heavy cross-striations and ring shadows, I looked into the matter of whether or not the patient had received phosphorus in oil, and received word that he had been a healthy breast-fed baby and had received no medication of any kind for any length of time. Dr. Rigler has demonstrated similar findings in scurvy in infants. It is possible that this boy may have had repeated mild attacks of scurvy as an infant. It is also possible that he may have had repeated mild attacks of lead poisoning.

RADIATION THERAPY OF DIPHTHERIA CARRIERS[1]

By IRA I. KAPLAN, B.Sc., M.D., Director, Division of Cancer, Department of Hospitals; Director, Radiation Therapy Department, Bellevue Hospital; Associate Attending Radiologist, Lenox Hill Hospital, *New York City*

IN the daily press several months ago, Commissioner Rice, of the New York City Department of Health, pointed out the necessity of guarding against a rise in diphtheria cases which would follow a letting-down of preventive measures. The Department has made a study of the cyclic recurrence of diphtheria and believes that a reduction in the number of diphtheria cases through immunization therapy may perhaps avoid the rise due to occur according to the cyclic trend. The Department warned against the expected higher incidence of diphtheria in 1940 and 1941, which is the period indicated in the cycle of recurrence when this disease has always been most prevalent.

Accordingly, greater attention must be paid to diphtheria prevention. While such preventive measures as immunization play an important part in the public health program for the control of diphtheria, it is not so generally known that diphtheria carriers disseminating the disease may be undoing much of our carefully planned work toward diphtheria control.

How to handle this question is an important public health problem, and it is a vital function of municipal health agencies to reduce to a minimum the number of diphtheria carriers.

In New York City, for the year of 1938, the Board of Health report showed 396 carriers on record, and during the same period 700 cases of diphtheria were reported.

During 1937, there were 121 diphtheria carriers under custody at the Willard Parker Hospital, New York City, who were confined there on an average of 19.9 days each. In 1938, of the 60 diphtheria carriers admitted to Willard Parker Hospital, four were persistent carriers and the average stay of all was 16.1 days. During the first six months of 1939, there were 34 cases of carriers at this hospital, for an average stay of 17.6 days.

During the first seven months of 1939, there were 389 cases of diphtheria in New York City, reported to the Board of Health and 222 carriers were registered. According to Anders, the disease is kept alive in a community largely by virulent organisms in immune persons. Since, unfortunately, no toxic symptoms are manifested by the carrier, his discovery is rendered difficult. Rush, Miller, and Perkins define a carrier as a person who harbors virulent diphtheria bacilli for a period of 21 days or more after an attack. They found the average duration of carrier condition to be 31 days.

As for the treatment of diphtheria carriers, Osler and McRae state that no satisfactory means have been found of ridding the carriers of bacilli. Various methods have been suggested, among them painting the throat with iodine or formaldehyde solutions and sprays or other antiseptics. In some cases spraying the throat with pure cultures of the *Staphylococcus aureus* was employed. Tonsillectomy and adenoidectomy have been tried; removal of foreign bodies and carious bones have been employed but without results. Antitoxin has been tried, but it has no direct action on the bacilli. The best treatment is that which can be easily administered without distress to the patient.

With the advent of the x-ray, it was suggested that in view of its potent biologic effects on the body it might also be bactericidal. All types of bacilli were treated with x-ray. Berton, carrying on research along these lines, experimented with the diphtheria (Klebs-Löffler) type, exposing

[1] Read before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

cultures of this to direct x-ray effect for as much as 64 hours. The effect as shown by guinea-pig inoculation proved these bacilli innocuous to x-ray.

In 1922, Hickey reported the results of treatment of diphtheria-carrier patients in Detroit, Michigan, with x-ray therapy. In his series, 19 involved the throat, 6 the nose, and 9 were associated with otitis media. The cures were:

Throat	15
Nose	4
Ear	4

Ledoux-Lebard reported treating 9 cases with good results. In 1932, Wahl reported similar good results from such treatment in the cases under his care. Dubowyi reported satisfactory results in 75 cases which he treated.

In 1938, Streil summarized his results in the treatment of 150 cases of diphtheria carriers with roentgen rays. His material comprised two groups. The first, the "dischargers," or those who, though recovered from the clinical form of diphtheria, still showed positive smears for from three to four weeks after the onset of the disease. The second group were the "accidentally discovered diphtheria carriers." Following x-ray treatment, good results were achieved in 95 per cent of the cases in the first group and in 97 per cent of the cases in the second group.

The action of the x-rays as already known is not directly bactericidal but experience has shown that irradiation favorably affects inflammatory conditions, the endotoxins formed thereby destroying the bacteria. In addition, fibrosis later sets in with shrinkage of tonsillar tissue, thus eliminating the crypt formation which is so favorable for the lodgment of diphtheria bacteria.

Heineke, and later Warthin, showed experimentally that as early as fifteen minutes after roentgen treatment lymphocytes undergo nuclear disintegration, which continues for several days. Desjardins and Hodges associated this rapid destruction of lymphocytes with the liberation of large quantities of labile, active endotoxins which are bactericidal.

Streil suggests that Pordes' therapy regarding the action of x-rays is most plausible. Pordes maintains that the beneficial action of x-rays is due to the mobilization of proteolytic ferments within the leukocytes. Under the influence of the x-rays these ferments exert the bactericidal action.

During the past two years we have been called upon to treat 11 cases of diphtheria carriers from the New York City hospitals.

There were five females and six males. The oldest patient treated was a female 63 years of age, the youngest was a 13-year-old girl. One of the women was a maid in a large private hospital when her condition was discovered. The oldest male patient was 27 and the youngest a boy 15 years of age. In most cases the nose and throat comprised the seat of the involvement, in one the disease was limited to the larynx, in a few cases only the throat was involved.

The duration of the status as a diphtheria carrier, as reported on admission, was from two to eight weeks. Cure was achieved in most instances within a few days following the last treatment. In most cases three treatments were required, in others four or five were needed to achieve results. In four cases, only one treatment was administered. In only two cases were untoward symptoms noted following x-ray therapy. These were manifested by pains in the jaws and neck, but passed away within the next day without active treatment. All these cases were treated by x-ray therapy according to the following technic.

High voltage x-ray therapy was employed, from 180 to 200 kv., with 0.5 mm. Cu plus 1 mm. Al filter, at from 40 to 50 cm. distance, through 8 × 10 or 9 × 12 cm. portals. Both sides of the neck were treated the same day and the rays directed through the tonsillar area. The dose was 150 r per treatment, per area, and three treatments were given one week apart. One week after completion of treatment the throat cultures are usually negative for

diphtheria. In some instances negative cultures may follow one or two treatments, in which case the third dose of irradiation may not be required. Occasionally some cases require a fourth treatment before a negative culture is achieved.

In our cases the treatment was immediately effective in 10 cases and delayed relief was obtained in one case.

If the diphtheria focus is in the nasopharynx, irradiation is administered directly through the nose and face area. The technic employed is similar to that used for the tonsillar treatment.

The results achieved are reported upon, and, through the courtesy of Dr. D. M. Tolle, Superintendent, Willard Parker Hospital, we are able to present the details of some of the cases treated.

A. F., aged 20 years, admitted to the contagious disease hospital on June 23, 1938, with the diagnosis of tonsillitis and presumptive diphtheria carrier, was found to be suffering from clinical diphtheria. Throat cultures were persistently positive. She was sent to Bellevue Hospital on Aug. 10, 1938, for x-ray treatment. Throat cultures were still positive but virulence test reported on Aug. 11 revealed the Klebs-Löffler bacilli were non-toxic. She was discharged on Aug. 13, 1938. Final diagnosis was tonsillar diphtheria and diphtheria carrier.

E. F., aged 63 years, was admitted to the contagious disease hospital on Sept. 27, 1938, and found to be suffering from tonsillar diphtheria. The patient's nose and throat cultures were persistently positive and virulence tests were toxic. She was sent to Bellevue Hospital for x-ray treatment on Oct. 31. The following two sets of nose and throat cultures were negative. She was discharged on Nov. 3, 1938.

Due to the contagious character of these diphtheria carrier patients, all necessary precautionary measures were taken to prevent contagion during the course of the radiation therapy.

M. B., 27 years of age, employed as a maid in another large hospital, was admitted to the contagious disease hospital on Oct. 16, 1937, with the diagnosis of diphtheria carrier—positive virulence. On admission, clinical examination was negative. Daily throat cultures were taken and these were positive, on Oct. 16 to 23, inclusive. A negative report was received on Oct. 24. The reports were again positive for Klebs-Löffler bacilli from Oct. 25 to 28, inclusive. Virulence examinations were reported as toxic on Oct. 16 and 22. During hospitalization, treatment consisted of isolation and throat sprays twice daily with gentian violet solution, without relief. On Oct. 28, the patient was given x-ray treatment at Bellevue Hospital.

TABLE I.—DIPHTHERIA CARRIERS TREATED WITH X-RAY

Patient	Age	Sex	Admission Date	Situation of Disease	Duration	Dates Treated	No. Treatments	Results Negative Culture
W. L.	26	M	Jan. 4, 1937	Throat	2 Months	Jan. 11, 18, 25, 1937	3	Feb. 16, 1937
E. S.	30	M	Jan. 11, 1937	Throat	1 Month	Jan. 11, 18, 25, 1937	3	Feb. 16, 1937
M. R.	22	M	April 27, 1937	Nose and throat	3 Weeks	May 3, 10, 17, 1937	3	May 21, 1937
A. N.	27	M	May 3, 1937	Nose and throat	1 Month	May 3, 10, 17, 24, 1937	4	May 31, 1937
M. B.	27	F	Oct. 28, 1937	Larynx	3 Weeks	Oct. 28, 1937	1	Oct. 29, 1937
A. B.	15	M	Feb. 23, 1938	Throat		Feb. 23, 1937	1	Feb. 23, 1938
A. F.	20	F	Aug. 10, 1938	Throat	10 Weeks	Aug. 10, 1938	1	Aug. 13, 1938
E. F.	63	F	Oct. 31, 1938	Throat	1 Month	Oct. 31, 1938	1	Nov. 3, 1938
M. S.	25	F	June 9, 1939	Throat	2 Weeks	June 9, 12, 14, 19, 1939	4	June 19, 1939
H. S.	26	F	July 26, 1939	Nose and throat	3 Weeks	July 26, and Aug. 2, 1939	2	Aug. 4, 1939
A. P.	13	F	July 26, 1939	Nose and throat	2 Months	July 26, and Aug. 2, 1939	2	July 31, 1939

That night she complained of excessive throat dryness and the following day she complained of pain in the muscles of the neck and pain in both ears. These painful sensations lasted 36 hours and were ascribed to the x-ray therapy. Throat cultures taken on Oct. 29 and 30 and on Nov. 1 were reported as negative for Klebs-Löffler bacilli and the patient was discharged cured on Nov. 2, 1937.

M. S., aged 25 years, was admitted to the contagious disease hospital on May 20, 1939, with the diagnosis of clinical diphtheria, involving the tonsils. Throat cultures taken daily from May 20 to 31, inclusive, were positive for Klebs-Löffler bacilli and the culture on May 20 was reported as virulent. Treatment consisted of the intramuscular injection of 10,000 units of diphtheria antitoxin and throat irrigations. The irrigations were discontinued after three days and the clinical condition cleared up. Throat cultures were continued daily from June 2 to 10, inclusive, and all were positive for Klebs-Löffler bacilli. On June 12, there was a negative report but from June 15 to 18, inclusive, the reports were again positive. A virulence test was repeated on June 4 and that also was found to be toxic. On June 9, 12, 14, and 19, the patient received x-ray therapy at Bellevue Hospital. On June 19 and 20 the throat cultures were negative and the patient was discharged June 22, 1939.

Local throat treatment during the "carrier" stage consisted of gentian violet throat spray twice daily, from May 24 to June 2, when lactic acid, 2 per cent solution, was substituted as a throat swab and was continued twice daily until the day of discharge. The night following the first x-ray treatment, the patient complained of pain in the jaw, after the second treatment she had a severe headache and was nauseated, refusing breakfast the following morning. After the third treatment there were no complaints, but the night following the fourth treatment she again complained of nausea, soreness in the throat, and pain on swallowing. In this case, as in the previous one, the uncomfortable sensations following x-ray treatment cleared up in from about 24 to 36 hours.

Table I shows the data of all the cases treated.

Conclusion.—X-ray therapy is a harmless effective method of treatment for persistent diphtheria carriers. It may be employed in all cases, regardless of age or sex. High voltage x-ray is employed. X-ray treatment was used only when all other forms of therapy proved ineffective.

755 Park Avenue.

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DISCUSSION

Rollin H. Stevens, M.D. (Detroit, Mich.): Dr. Kaplan's paper is very timely. I have no personal experience of importance to add.

In 1922, when Dr. Hickey was doing his work, we treated a number of cases of diphtheria carriers with apparent success, but have not had any cases referred to us for a long time. In Detroit, immunization is carried out quite extensively, and we do not hear so much about diphtheria.

There must, however, be a few carriers. How or where they are treated I do not know, as we are not connected in any way with the Board of Health.

However, we have had enough experience in the x-ray treatment of infections in general, and a few diphtheria carriers in particular, to warrant our endorsement of x-ray therapy for diphtheria carriers.

The original work, showing a high percentage of cures, however, was done with low voltage therapy, about 85 or 90 kv. The tissue dose to the mucous membrane was small, and that can be as easily accomplished by low as by high voltage, at a moderate rate of delivery.

Dr. Kaplan's paper should serve as a notification to the health departments of the country that in the process of immunization against diphtheria, x-ray therapy of carriers is the simplest and most effective method known to render carriers safe to mingle with the hosts of persons with whom they may come in contact and who may not be immunized.

The Klebs-Löffler bacillus may exist in a throat the soil of which, at the moment, may not be of the proper character to make it virulent, and so the disease does not spread until the period in the cycle Dr. Kaplan mentioned, when the bacilli seem to become virulent again and start an epidemic.

In these periods of low virulency, we are apt to grow lax, and the disease gains a new foothold.

According to recent investigations, the mucin in the mucous membrane protects the germs. Irritating antiseptics increase the flow of mucous by irritating the mucous membrane, and thus serve to protect the infectious organisms. Therefore, the use of strong antiseptics on the mucous membrane would seem to be contra-indicated.

On the contrary, two or three mild doses of x-ray in some way seem to make the living tissues unfavorable for the growth of noxious germs, and do not irritate the mucous membrane or cause any harm.

There are many theories as to the mode of this accomplishment advanced in the literature of x-ray therapy of infections, but the truth is not yet known.

WALTER C. POPP, M.D. (Rochester, Minn.): I deem it a great favor to be permitted to discuss Dr. Kaplan's excellent paper. I feel that he has brought to our attention again the question of diphtheria carriers by showing in his case reports certain very important features which are essential in considering a diphtheria carrier as cured. The first is the fact that not all diphtheroid bacilli are pathogenic. This necessitates a virulence test to determine whether or not the organism should warrant consideration insofar as carrier possibilities are concerned. The second is that more than one culture is necessary to consider a carrier of diphtheroid organisms as cured. The majority of public health bureaus insist on at least three successive negative cultures to consider a carrier as free of diphtheroid bacilli. I think these two points are quite essential and should be kept in mind when a carrier comes to the radiologist for treatment.

Our experience with diphtheria carriers has been limited because very few patients with this condition are seen at the Mayo Clinic. Those who have been treated have yielded negative cultures and were no longer considered to be carriers. We have treated our patients with voltages generated at 135 kv., using, in general, the same dosage as that suggested by Dr. Kaplan.

The ease with which roentgen treatment can be given makes it a convenient and safe method. The certainty with which a cure can be obtained in the majority of patients tends to make roentgen therapy the method of choice in treating diphtheria carriers. I believe we have in our hands an agent which is of great assistance to public health authorities in the control of diphtheria epidemics, provided it is used only after the closest co-operation of recognized bacteriologists and radiologists.

PRE-OPERATIVE IRRADIATION IN CARCINOMA OF THE BREAST[1]

A HISTOLOGIC STUDY

By EDMUND P. HALLEY, M.D., Department of Radiology, St. Joseph's Hospital, *Stockton, California, and* PERRY J. MELNICK, M.D., PH.D., F.A.C.P., Division of Cancer Diagnosis, Illinois State Department of Public Health; and Department of Pathology, University of Illinois College of Medicine, *Chicago*

RADIATION pathology, *i.e.*, the histologic mechanisms by which radiation destroys tumors, is of great practical importance to radiologists, who need to know what happens in a tumor when it is irradiated. Such knowledge is at present incomplete.

In contrast to the inflammatory diseases, the pathologic anatomy of tumors (aside from the recent developments along endocrine and chemical lines) has been largely concerned with descriptive morphology and classification. Tumor pathology furnishes information of great value to both surgeon and radiologist, yet it consists chiefly of a collection of static pictures. Even attempts to determine histogenesis are often based on no more than a morphologic resemblance to this or that cell of origin; and attempts to determine the degree of malignancy or radiosensitivity from the histologic appearance are limited by as yet comparatively meager clinical statistics, or are largely intuitive judgments based on nothing more definite than the old law of Bergonie and Tribondeau, formulated in 1906.

When a tumor is irradiated a dynamic situation supervenes, and complex histologic changes take place. These changes should be familiar to pathologic anatomists, yet we find this field of knowledge crowded with theories, hypotheses, contradictions, and analogies from other biologic test material.

In reviewing the literature of the many histologic studies which have been made of irradiated tumors one is struck by the fact that the bulk of these studies were made following single large doses, *i.e.*, the massive dose method. Much of the work was done with radium, in which the large doses close to the applicator often introduced an element of damage to normal tissues which is difficult to evaluate. Specifically, histologic studies of tumors irradiated by the newer fractional and protracted methods are scarce. It may well be that we should revise our views on radiation changes in connection with fractional methods of irradiation.

The histologic changes that have been observed in irradiated tumors by the many workers in this field can be summarized in a general way, briefly, as follows:

First, it is widely accepted that after a latent period of a few days a great many tumor cells begin to degenerate; that is, they die off by a process of radiation necrosis. The details of this necrosis have been worked out by many, and especially well by Ludford (1).

A second type of histologic change has been generally observed in irradiated tumors, consisting of an inflammatory or reactive mechanism. Infiltrations of various kinds of leukocytes take place, as well as proliferation of fibrous connective tissue; and, also, obliteration of blood and lymph vessels in various ways. The view has become widely prevalent that radiation stimulates these natural reactive mechanisms of the body in combating tumor cells. This view has been especially emphasized by Ewing (2), who has frequently expressed the opinion that fibrosis and obliteration of blood vessels are at least equal in importance to the direct action of radiation on tumor cells. Armed with this con-

[1] Presented before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

cept, radiologists have felt that, on the one hand, they were destroying many tumor cells by depriving them of blood supply, and also locking them up, so to speak, in a dense scar tissue; and on the other hand, preventing their dissemination by obliterating the lymphatics and blood vessels in various ways.

A third type of histologic change has occasionally been described. This consists of a change in the character of some of the tumor cells. Often they become more adult in type, undergoing squamous metaplasia in squamous-cell carcinoma, or becoming simple benign-appearing gland-like cysts in adenocarcinoma. Often abnormal, bizarre, or giant forms of tumor cells are seen. Here and there the thought is expressed in the literature that such changes result from alteration of the tumor cells in some fundamental way, such as mutation-like changes produced by radiation. This concept was foreshadowed twenty years ago by Alter (3), and has more recently been discussed by Griffith and Zimmer(4)and by Crowther(5).

Although many clinical studies have been published, histologic studies of carcinomas of the breast after pre-operative irradiation are disappointingly few. There are scattered reports of a few cases by various authors, but the largest amount of material has come from the Memorial Hospital, New York City. In 1924, Lee and Herendeen (6) reported a number of irradiated breast cancers which showed the expected fibrosis and blood vessel changes. Adair and Stewart (7), in 1935, and Adair (8), in 1936, reported a large number of cases treated by x-ray or by radium. Here the histologic changes are also described as being chiefly fibrosis and blood vessel changes.

This leads to a most important point in the management of irradiated breast cancers. On the basis of these concepts an interval of at least two to three months has become widely accepted as the period which must elapse, after irradiation is completed, before surgery may be done. This view can be summarized essentially by the following quotation from Adair:

"It is necessary to allow two months as a minimum after the completion of the treatment, for irradiation to become fully effective in producing the desired tissue changes. The direct killing effect on the more sensitive cancer cells, together with the slower development of fibrosis and endarteritis and other results of irradiation, consumes a period of at least eight to ten weeks.'

Unfortunately, such a prolonged pre-operative wait makes both patient and surgeon restless. When radiation is carried to top limits of tissue tolerance, as in a series recently reported by Adair (9), the production of tumor-bed fibrosis leads too often to serious wound repair problems. Most radiologists have been reluctant to carry irradiation quite so far. Further, the blame sometimes attached to radiation therapy by less competent surgeons for poor, healing really due to surgical errors, has decreased the use of this modality.

An approach to the problem which avoids the long interval between diagnosis and the solace of mastectomy is the procedure so ably championed by Pfahler (10). A short pre-operative treatment given within from ten days to two weeks is followed immediately by operation, before skin and tumor-bed reaction appears. Thorough post-operative irradiation follows promptly. It is argued that such pre-operative irradiation devitalizes the more malignant cells and exerts an effect on normal tissue detrimental to implantation of carcinoma. Occasionally, one hears that the lymphatics tend to be sealed off even with these lesser doses.

The histologic evidence to be presented here argues strongly for revision of these concepts. The first method entails too long an interval between irradiation and operation as viable tumor cells by then have resumed their growth, invasion, and spread. The second method fails to achieve optimal tumor-cell damage beyond primary radionecrosis of the few most radiosensitive cells.

Our findings in irradiated carcinoma of the human breast are a sequel to, and dependent upon, the studies of transplantable rat tumors treated with x-ray and re-

ported, in 1937, by Melnick and Bachem (11). In these studies, the results of greatest interest were the histologic findings in those tumors treated by various forms of protracted and fractional radiation.

Summarized briefly, the essential findings in about 300 transplantable rat tumors irradiated by various methods are as follows:

After massive dose methods, the classical picture outlined above is seen, namely, radiation necrosis of tumor cells, associated with hyperemia, cellular infiltration, granulation tissue proliferation, and, finally, fibrosis and blood vessel changes.

After fractional and protracted methods of irradiation, however, an entirely different picture is seen. The most radiosensitive cells are, it is true, destroyed by necrosis; but the remaining resistant cells are seen to undergo a different change consisting apparently of a mutation-like alteration in their character. The small daily doses transform them into pleomorphic abnormal forms, giant tumor cells. This giant-cell formation results from the direct action of the radiation on the tumor cells, not from secondary reactive mechanisms. Furthermore, these giant cells do not degenerate by necrosis, but by calcium deposition in their nuclei. It is logical to conclude that these abnormal cell forms are mutation-like changes resulting from the progressive cumulative action of the small daily doses.

These small daily doses at the same time have no apparent effect on the adjacent normal tissues. There is no fibrosis and no blood vessel damage. Normal tissues recover from the small doses. Dividing tumor cells cumulate mutation-like, irreversible, radiation changes in their nuclei. Much of the fibrosis and blood vessel obliteration seen in irradiated tissues obviously result from more or less damage to normal structures by irradiation at an intensity too high, or with individual or total doses too large, to allow normal tissues to recover: 1,500 r applied to a rat tumor in one dose will produce fibrosis and blood vessel change. However, 100 r daily for 15 or even 30 days will not. These experiments with x-radiation have recently been repeated using radon implants and were reported by Melnick (12), at the Third International Cancer Congress. The results parallel the earlier study in a gratifying manner.

Human material for the study of irradiation changes is not abundant. Even serial biopsies of accessible tumors give us only isolated stages, like single frames from a motion picture in which the poses of the actors have only a limited meaning. On the other hand, transplantable animal tumors, as used in the above experiments by Melnick and Bachem, furnish a uniform, easily controlled material for this kind of study, in which the changes can be followed closely day by day. If the isolated stages obtained from human material closely match animal material at corresponding treatment levels, a basis for reconstructing the mechanisms involved in irradiated human cancer would seem justified.

In the usual case of carcinoma of the breast amputated from two to three months after the end of irradiation, it is surprising to find how much intact and uninjured cancer is seen in the great majority of cases. Kress (13) reports only 9 per cent completely destroyed in a large series of cases. Adair reports as high as 33 per cent completely destroyed; however, the technic of aspiration biopsy probably detracts from the strict scientific value of this figure. The finding of large amounts of intact cancer, commonly seen at from 8 to 12 weeks after irradiation, has generally been interpreted by pathologists as tumor tissue which has escaped the action of the radiation.

In view of the experimental findings in animal tumors outlined above, the thought naturally occurred that perhaps we are missing the actual irradiation changes, which occur earlier. Accordingly, earlier amputation was requested. Excellent cooperation from our surgeons made possible the accumulation during the past two and one-half years of 21 cases of breast cancer

in which tumor doses[2] range from 1,200 to 4,500 r, administered variously over periods of from 11 to 49 days. The interval to operation ranges from 1 to 45 days after the end of irradiation. In the cases receiving the larger total doses, treatment was carried to a full second-degree skin reaction with erythema, vesiculation, and desquamation. Except for the first few cases, surgery was undertaken as soon as desquamation was complete when the skin became smooth and dry, so that surgical cleanliness was feasible.

The histologic findings in these cases correlate closely with those found in animal tumors. In the early stages, up to the second week, necrosis of the radiosensitive tumor cells predominates. Case M. W. (Fig. 1-*A*), in which mastectomy followed by one day after about 1,600 r tumor dose delivered over 17 days, demonstrates these earlier changes in which vacuolation and pyknosis is found with hardly a trace of pleomorphism. Many intact tumor cells are still present.

That damage to the tumor cells beyond this simple necrosis has been effected is well illustrated by Case M. R. (Fig. 1-*B*), in which 1,200 r tumor dose was administered over a period of 11 days and excision deferred 17 days after the end of treatment. Many bizarre-shaped cells with irregular nuclei can be seen—pleomorphism. However, numbers of apparently normal cells are still present.

Beginning about three weeks after more extensive irradiation, in addition to the remnants of radiation necrosis, fully developed pleomorphism of the remaining tumor cells can be seen. This process is especially well illustrated by Case C. M. (Figs. 1-*C* and 1-*D*). Mastectomy was done 22 days after a tumor dose of 2,400 r delivered through two ports over a period of 34 days. The surviving cells are nearly all transformed into giant cells, many of whose nuclei are beginning to calcify. Furthermore, they have lost their invasive power, and are proliferating only along preformed channels, *i.e.*, the ducts and alveoli. Meanwhile, the tumor bed appears to be intact. The photomicrographs show extensive radiation changes in the surviving tumor cells, yet with intact blood vessels and even intact loose areolar tissue normally found about the lobules of breast glands; all this in a heavily irradiated field.

Case V. B. (Figs. 1-*E* and 1-*F*) illustrates the tremendous changes accumulated by protraction of radiation. This patient received a total of 3,400 r through two ports over 51 days, and operation was done 17 days later. Both photomicrographs are of equal magnification. The first is a biopsy before treatment; the second shows an almost complete transition to the large giant forms. Reaction of the tumor bed, as manifest by changes in the connective tissue, is negligible and abnormal vascular patterns are not found.

Four or five weeks after the end of irradiation, only the last remnants of irradiation change can be seen. Case B. N. (Figs. 1-*G* and 1-*H*) received a total of 2,600 r into the tumor area over a period of 32 days and amputation was done 29 days after the end of the treatment. Small clumps of débris containing groups of calcified giant-cell nuclei are found, some being phagocyted by foreign body giant cells. In addition, however, a startling resumption of activity of the surviving tumor cells has taken place. Intact, newly proliferating carcinoma simplex is present, with the appearance of normal looking carcinoma cells along lymph channels, evidence of resumption of invasive power.

Clinical verification of regrowth is oftimes present. In one case in which the patient received about 2,300 r tumor dose over a period of 33 days, the tumor shrank from about 3 cm. diameter to a barely palpable node. At the end of four and one-half weeks the tumor was obviously increasing

[2] Tumor dose has been estimated with the aid of Quimby's tables ("Data Concerning Tissue Dosage," Radiological Society of North America, Refresher Course, Annual Meeting, Pittsburgh, December, 1938). This was done for simplification, since therapy was carried out independently by two radiologists. Equipment changes during this period resulted in employment of a wide range of radiation quality. No correlation between quality of ray and degree of histologic change was found.

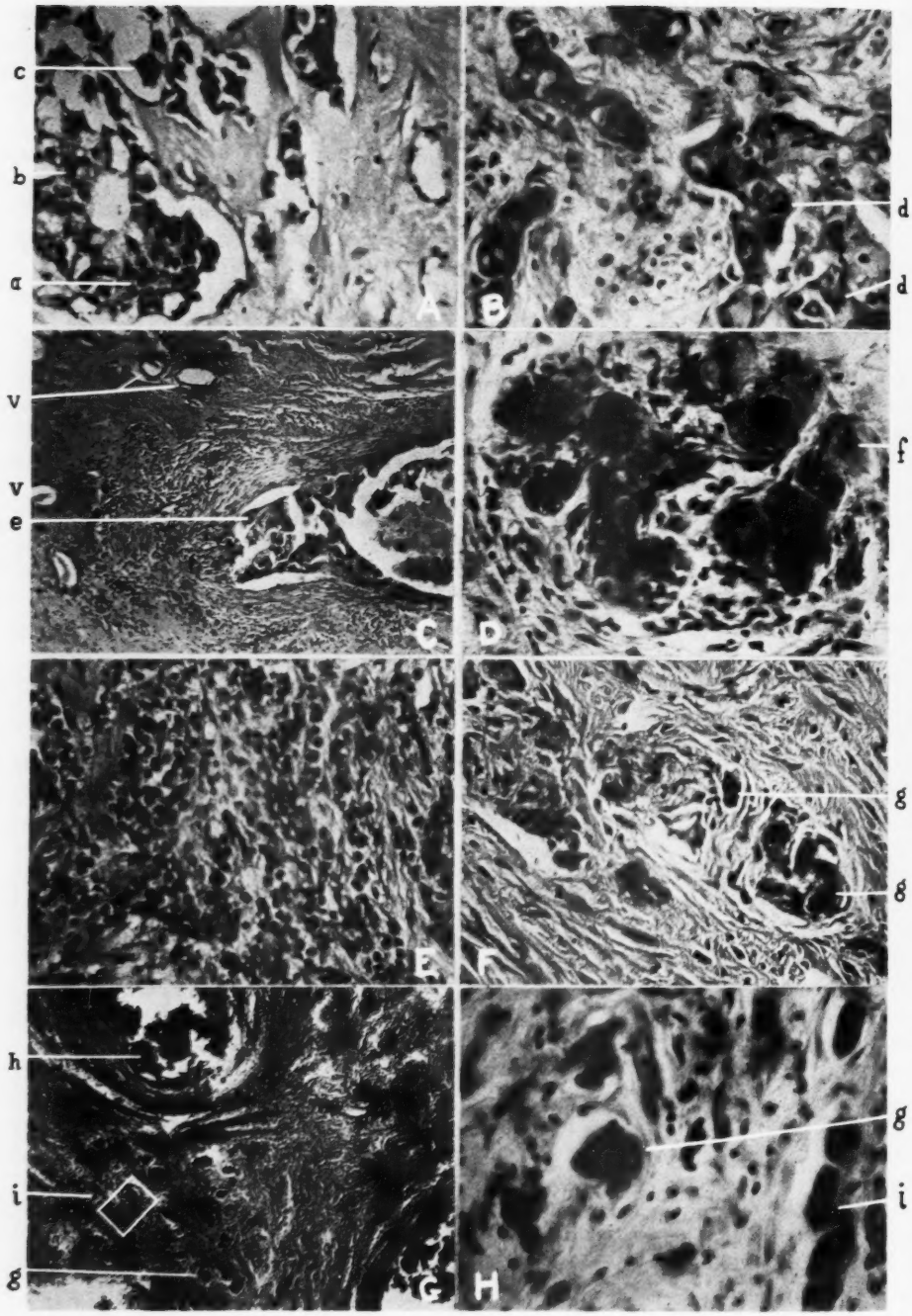

Fig. 1.

in size and at six weeks, just before excision, it was nearly 2 cm. in diameter. The sections in this case showed an abundance of normal-appearing tumor cells with only a few late degenerative forms.

The picture at 8 to 12 weeks is usually that of intact carcinoma, which has, in reality, been growing for from one to two months, in spite of the fibrosis that has sometimes developed by about that time. It is probable that an active, viable cancer cell has very little respect for even the densest fibrous tissue.

The parallel between these changes in human carcinoma and those demonstrated in experimental animals, by Melnick and Bachem, may be seen by comparing photomicrographs in Figure 1 with those in Figure 2, which has been borrowed from their material. Figure 2-*A* is from an untreated control, Flexner-Jobling rat carcinoma (strain 223–I). Figure 2-*B* shows

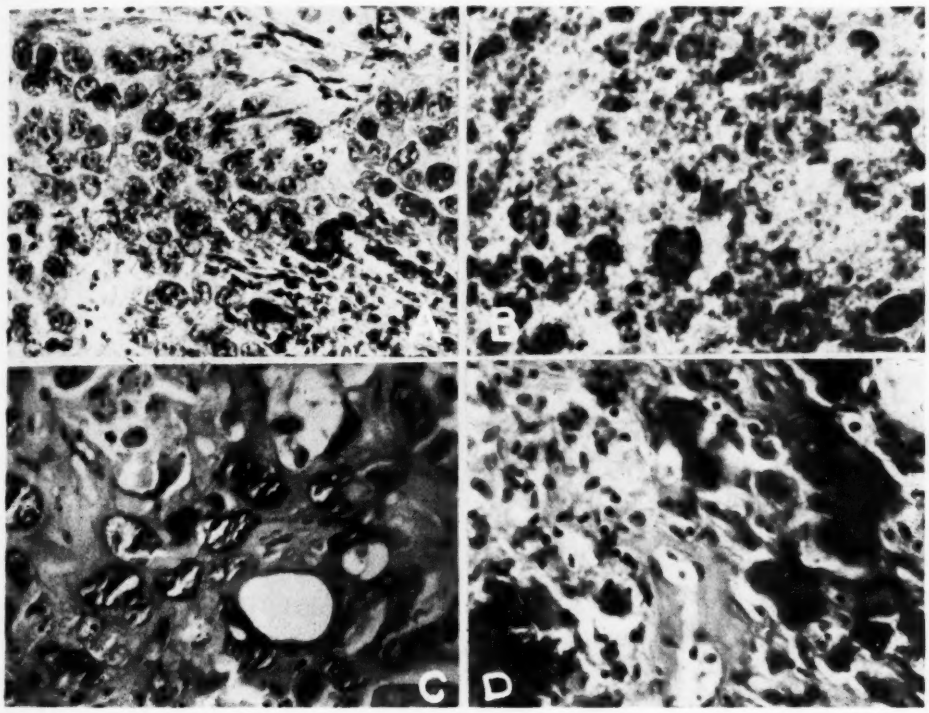

Fig. 2.

See preceding page for illustration.

Fig. 1. Human breast carcinoma. (Dosages are estimated tumor dose.) *A*, Case M. W., given 1,600 r in 17 days; mastectomy one day later. Primary radiation effects. (*a*) Intact tumor cells; (*b*) vacuolation; (*c*) pyknosis; × 200. *B*, Case M. R., received 1,200 r in 12 days; mastectomy 17 days later. (*d*) Early pleomorphism; × 200. Mutation-like changes continue to develop after treatment is terminated. *C*, Case C. M., given 2,400 r in 34 days; mastectomy 22 days later. (*e*) Advanced pleomorphism with loss of invasive power; (*v*) intact blood vessels; × 100. *D*, same as *C*. High power from an adjacent field. Pleomorphic giant cells. (*f*) Cell nucleus about size of an untreated carcinoma cell; × 400. *E*, Case V. B. Biopsy before treatment, carcinoma simplex; × 200. *F*, same as *E*, after radiation. Total dose of 3,400 r in 51 days; mastectomy 17 days later. (*g*) Advanced pleomorphic giant cells. (Note that magnification is the same for these two sections.) *G*, Case B. N., received 2,600 r in 32 days; mastectomy 29 days later. (*g*) Advanced pleomorphism; (*h*) calcareous débris; (*i*) regrowth along lymphatic channel; × 70. *H*, High power of area indicated by rectangle in *G*. (*g*) Advanced pleomorphic giant cell; (*i*) regrowing carcinoma cells; × 400.

Fig. 2. Flexner-Jobling Rat Carcinoma (from Melnick and Bachem). *A*, Untreated control; × 300. (These four sections are of same magnification.) *B*, Primary necrosis; six days after administration of 1,000 r in a single dose. *C*, Pleomorphism; 150 r daily for eight days. *D*, Calcified giant cells; 150 r daily for 12 days.

primary radiation necrosis six days after a single dose of 1,000 r, and presents the classical picture of radiation effects. Figure 2-*C* is from a tumor given 150 r daily for eight days; early pleomorphism and beginning giant-cell formation is present. Figure 2-*D* shows the accumulated effects of 150 r daily for 12 days. Giant cells have undergone extensive calcification of their nuclei. Keeping in mind that the cycle of growth and reaction is much shorter in the rat than in the human, and that larger daily doses (comparatively and actually) can be given to the experimental animal than are clinically feasible for the human, the similarity of reaction to treatment is surprisingly close.

From this histologic evidence it is logical to conclude that radiation by fractional methods acts directly upon human breast tumor cells in a twofold manner, producing early a simple radionecrosis of sensitive cells, and later by accumulation of mutation-like effects through succeeding generations of daughter cells. This latter may easily be the more significant mechanism. Damage to the tumor bed, as manifest by fibrosis, and by blood and lymph vessel obliteration, does not occur under usual fractional radiation. Further, regrowth of surviving tumor cells appears four to six weeks after termination of therapy.

If this be true, it would seem that the use of a short pre-operative course of irradiation fails to achieve an optimal tumor damage, as the mutation-like effects require from four to five weeks of radiation. Since tumor-bed damage is slight under fractional methods, this mutational mechanism can well be followed to a full second-degree skin reaction. Surgery done within two or three weeks of the end of irradiation has the advantage of a tissue in which viable tumor cells have been decreased to a numerical minimum. Delay beyond four weeks faces regrowth of the tumor, and after from 8 to 12 weeks the operation is being done, with the tumor almost identical in character with that of the preradiation neoplasm.

As for surgery closely following an acute skin reaction, and through an erythema, there have been no complications in this series. Before early operation was undertaken, we irradiated a number of rabbits by fractional methods comparable in manner and total dosage to the human procedures. Treatment was given over one rump only to minimize systemic effects and to preserve the opposite rump for control. At variable intervals after the end of treatment, an elliptical excision was made in the treated area and duplicated on the untreated rump. Healing was invariably as good, and in most animals better, on the treated side as compared with the untreated. In this human material, the surgeons have frequently expressed the opinion that healing was more prompt in the irradiated patients. After all, erythema means increased blood supply; hence, one should expect better healing.

SUMMARY

The histologic findings in a series of human breast cancers irradiated by fractional methods is presented. Amputation of these breasts was performed earlier than the usually recommended interval of from two to three months, namely, from two to four weeks after the end of irradiation to a full second-degree desquamative skin reaction.

The principles of radiation effect under fractional methods, as previously elaborated by Melnick and Bachem for experimental animal tumors, are found to apply in this human material. Sensitive tumor cells under fractional irradiation undergo primary necrosis. More refractory tumor cells, by damage to their chromosomal structure, develop abnormal mutation-like forms (giant cells) which fail to survive and frequently degenerate by calcification of their nuclei. Normal tissues, including lymph and blood vessels, were found intact. Fibrosis was not seen following fractional treatments. Therefore, it is logical to give adequate pre-operative irradiation over at least four or five weeks in order to reduce the viable tumor cells to a numerical minimum.

About four weeks after the end of irradiation the carcinoma begins to proliferate again from the surviving tumor cells, which remain in at least 90 per cent of irradiated breast cancers. Since with fractional methods obliteration of blood and lymph vessels does not occur, it is imperative to amputate early before the tumor resumes its growth and returns to its preradiation status, with danger of invasion and metastasis.

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DISCUSSION

GEORGE E. PFAHLER, M.D. (Philadelphia): I have been interested in pre-operative irradiation for at least twenty-five years. I have been talking about it; and I have been using it. I am quite sure that the value of pre-operative irradiation is not thoroughly appreciated either by radiologists or surgeons.

I think Dr. Halley has given us a real contribution to-day in demonstrating histologically these changes which do occur pre-operatively or, at least, in the tissues which we prepare for operation.

He is the first really to attack this problem in this manner. Not that he is the first to have made histologic studies following irradiation, but he has studied the effects from irradiation such as we use in our daily work.

I have never used the so-called massive dose treatment. I have always, beginning forty years ago, used the fractional dose method of treatment: at first, because I was afraid to do anything else; and then, gradually, having produced results, I followed the same general rule.

I have read the article by Dr. Melnick and Dr. Bachem, and I certainly recommend that every one of you read it. Furthermore, I have pleaded with Dr. Melnick to present his studies before some radiological body so that, instead of finding it published in a pathological journal, you will find it published in the radiological journals, in language we can understand.

It was a great satisfaction to me to find that Dr. Melnick proved by his investigations that the saturation method produces better results than any of the others. Dr. Juul, of Copenhagen, had proved the same sort of thing. Dr. Reisner, in Holfelder's Clinic, did some excellent experimental work along that line.

I have not been talking much about the saturation method lately because it does not differ greatly in its practical application from the Coutard method. In cases in which it is possible to apply a large dose at the beginning of treatment and then follow with sub-doses, it does not differ from what Coutard is doing. But in practical work, such as treatment of the breast, for example, it is impossible to do it by that method: we must use fractional doses. The patient becomes too sick from massive doses, and too much damage is done to the tissues. So, from a

practical standpoint, as I argued with Dr. Coutard many years ago, there is little real difference in the way we do our work in practical daily use of the rays.

With regard to the application of this problem in carcinoma of the breast, Dr. Halley has shown you beautifully these results; and he has shown that it is best to wait for a time longer than the week following the administration of pre-operative treatment in order to get the greatest effect. That is true when you come to study the tissues microscopically, but the actual damage to those cells has been done before you can see them microscopically, so I do not believe that it is an argument for waiting longer after we have given the pre-operative radiation.

In dealing with carcinoma of the breast, we have to adapt ourselves to circumstances, but I aim to give as a routine the pre-operative treatment necessary in about twelve treatments, which takes about two weeks. If I sterilize the patient at the same time as I do when treating a patient still in the menstrual life, then I allow three weeks in which to administer preliminary treatment since I want to treat ovaries as well. Then, during the following week, at the practical convenience of the surgeon and the patient, that patient can be operated upon without any extra risks so far as the patient is concerned.

The dosage given is between 900 and 1,200 r units. That will not destroy cancer. It is not as much radiation as Dr. Halley has shown you, but, I am aiming to destroy only those more malignant cells—those are more sensitive to radiation because they are the cells which cause surgical failures. We are willing for the surgeon to take away those that are less sensitive, and he will have success. If we can destroy the more malignant cells and bring this grade of cells down probably to Grade I or II, the surgical results will be approximately doubled. That is what my five-year recovery record shows—about 55 per cent recovery in the cases with axillary involvement.

That is about double what you can get with surgery alone and, therefore, from a clinical standpoint, it is quite worth while. I am a clinician. Dr. Halley, you must not forget, was basically a clinician trained in pathology—a pathologist—and he, fortunately for our sakes, has taken up this work in radiology, and is now teaching us some of the things which we should know.

ROENTGEN FINDINGS IN CAISSON DISEASE OF BONE, WITH CASE REPORTS[1]

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THE purpose of this communication is to add further description of caisson disease of bone to the small number (about seventeen) already reported, in order to emphasize its rarity and the characteristic features which may facilitate roentgen recognition. A full discussion is unnecessary in view of the recent excellent review of aseptic necrosis of bone by Kahlstrom, Burton, and Phemister (1), in which were presented twelve cases of caisson disease of bone from the literature and four additional cases from the authors' personal experience. The reader is, therefore, referred to this article for detailed study of the pathology, especially in the case in which autopsy was performed.

For the sake of clarity, the following summary of the history and pathogenesis of caisson disease is derived from their paper. In 1871, Bert defined caisson disease as the injury to tissues subsequent to the too-rapid removal from the decompression chamber, due to bubbling of nitrogen in the blood. Since fats dissolve more than five times as much nitrogen as an equal volume of water or blood plasma, fat or lipoid tissues suffer the most damage. In 1888, Twynam reported a case of caisson disease which developed massive necrosis of the shaft of the femur, with secondary pyogenic osteomyelitis. In 1911, six cases were described (Bassoe; Bornstein and Plate), involving hips or shoulders, usually the former. Mottled changes were noted in density in the head of the femur, with flattening of its articular surface, irregularity and slight narrowing of the cartilage space, and marginal lipping. Bassoe noted chronic changes in the cancellous bone away from the joints in three instances (upper diaphysis of tibia, lower diaphysis of tibia, and lower epiphysis of fibula). There were mottled areas of absorption and irregular lines of new bone formation, due to aseptic necrosis. Plate reported one case in 1928, and Christ, four, in 1934. Burkhardt, in 1934, classified caisson disease joint pathology under arthritis deformans.

Acute symptoms of caisson disease are referable to the spinal cord, internal ears, brain, subcutaneous tissue, and the limbs. Severe pains in the abdomen or limbs, hemorrhages in the skin and mucous membranes, impairment of hearing, and cerebral or pulmonary embolism may occur. Chronic lesions include pathologic changes of the bones and joints, paralysis from damage to the spinal cord, and impairment of hearing. In early cases the hips, less often the shoulders, knees, and ankles are painful. The joints usually clear up but in some instances deforming arthritis results.

The primary lesion is an accumulation of nitrogen gas in the bone (whether intra- or extra-vascular, or both, is a moot question), with a resultant massive aseptic necrosis. In the head of the femur or humerus, the necrotic lesions may break down and be invaded by, or eventually replaced by, new bone or fibrous tissue. Invasions of portions of the epiphysis may be followed by calcification and arrested transformation, especially in portions of the epiphysis away from the articular surface. Involvement of articular cortex and cartilage leads to slow development of arthritis deformans, with or without osteocartilaginous loose bodies. These changes resemble those seen after long interruption of blood supply, as in slipped epiphysis, fracture,

[1] Accepted for publication in June, 1939.

or dislocation. Among the four cases described by Kahlstrom, Burton, and Phemister were extensive lesions in the diaphysis and epiphysis of certain bones (femur, tibia, humerus), with changes in the joints. In the shafts there was an extensive necrotic area, 1 to 18 cm. in size, with a calcified and ossified zone of demarcation, the sequel to aseptic necrosis.

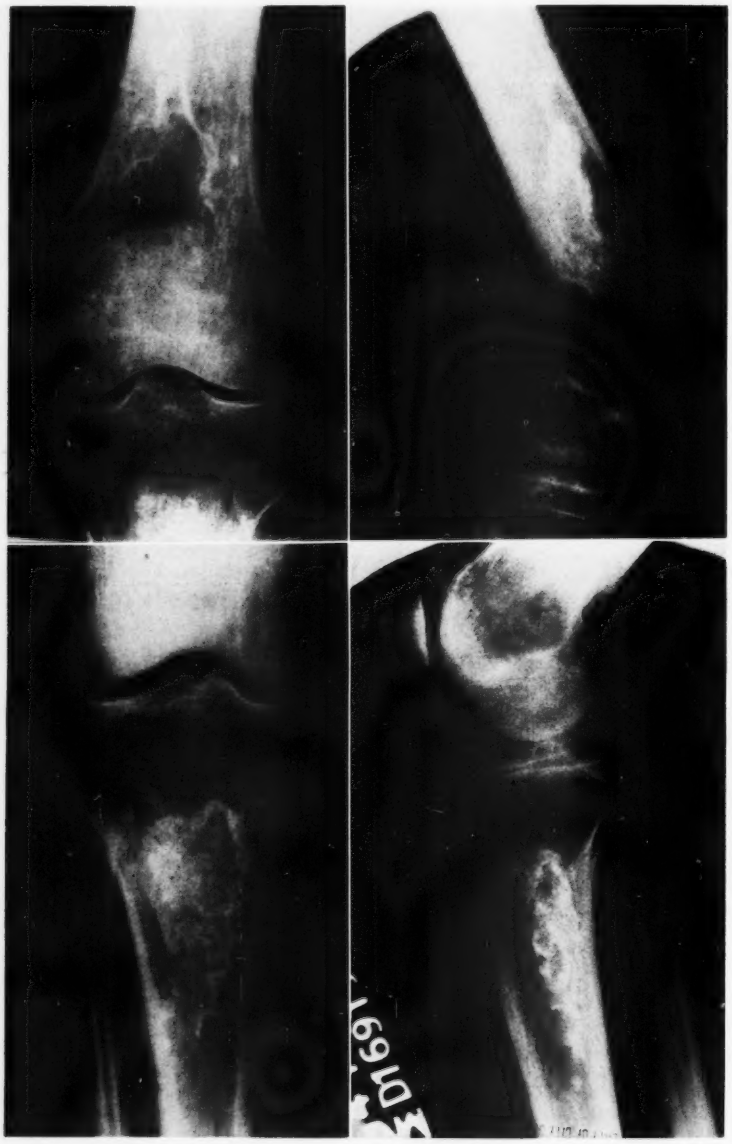

Fig. 1. Case 1, demonstrates the unusual calcification of the medullary canal in the diaphyses. Cortex and periosteum of the shaft are normal.

Frank and Knoflach (2) have recently reported a case of caisson disease which they call "divers' paralysis." In their report

films taken one and one-half years after the onset showed match-head-sized round rarefactions in the head of the femur.

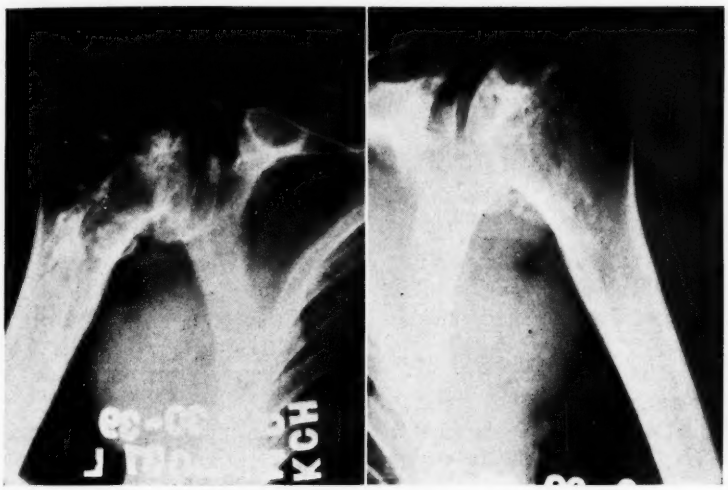

Fig. 2. Case 1. Necrotic areas in the humeral heads. Medullary calcification also present.

Five months later, shoulder films revealed a few 1-mm. wide straight lines of illumination separating portions of the cortex from the head of the humerus.

The unusual medullary calcification at the ends of long bones has been stressed as an important roentgen feature, in conjunction with the chronic bilateral osteo-arthritis of hips or shoulders or both. It seems advisable to emphasize that the necrosis in the head of the long bone, and the resultant osteo-arthritis, may be the only roentgen manifestation of caisson disease. We are presenting four cases that show: first, the typical medullary necrosis and calcification, and second, the type in which the characteristic calcification is absent.

Case 1. A. Y., Russian male, aged 48 years, admitted to the orthopedic service of Dr. Joseph B. L'Episcopo, Kings County Hospital, on Jan. 21, 1937, for treatment of osteo-arthritis of the hips and shoulders. The patient's story follows: He had been well until a few days after starting work on the construction of the Hudson Tube in 1916. His assignment was to assist in conveying muck in the electric trucks, work which was done under

Fig. 3. Case 1. The upper figure shows necrotic areas in the femoral heads, and the osteo-arthritis, before operation. The lower figure shows evidence of the left osteotomy.

45 pounds pressure. On one occasion the pressure was changed too quickly, and,

while approaching the surface, he experienced sudden pain in the hips, knees, and shoulders when about eight feet from the top of the elevator shaft. He was taken to a dressing-room and kept running around. Then he was replaced in the tank for a few minutes, with no relief of the pain. After a hypodermic, he slept. On awakening, the pain was somewhat relieved, but stiffness persisted in the extremities. Subsequent treatment for two months included medication, baths, and massage, with gradual reduction in the severity of the pain. In the intervening years the pain in the shoulders and hips has continued, and limping and stiffness have resulted.

Physical examination upon admission was negative except for the extremities. Upper extremities: no paralysis; movement at the joints is free; reflexes are hyperactive; some crackling at the shoulder joints on movement. Lower extremities: partial ankylosis of both hips; patient can flex right hip about 45 degrees, left hip about 40 degrees; internal and external rotation practically nil; knee joints appear normal; knee and ankle jerks hyperactive; Babinski negative.

A reconstruction operation of the left hip was performed by Dr. L'Episcopo on March 11, 1937. The operative findings were: "Capsule thickened and markedly adherent; motion almost completely restricted in the acetabulum; bone was hard but fragmented easily; flexion to 70 degrees and abduction 30 degrees; transverse wedge osteotomy done." The biopsy report after operation was chronic hypertrophic osteo-arthritis.

The patient was readmitted on Aug. 12, 1937, after convalescence in a rest home, emphasizing the serious economic ravage of this disease. Physical examination at this time: "Left hip, active flexion to 130 degrees, passively flexed to 90 degrees; motion slightly painful; good abduction and adduction on the left (side operated upon), none on the right; patient has improved and can now walk short distances; muscle therapy ordered."

He was admitted for the third time on Jan. 30, 1939, for further physical therapy. The patient has improved, with increased abduction, adduction, and flexion of the left hip. Motion in the right hip is found to be good. Further muscle therapy desired to develop the quadriceps and the ability to hyperextend the left knee on walking.

X-ray Examination.—A complete examination of the osseous system and the chest was made, summarized as follows:

Skull—no abnormal features.

Lungs and heart—slight thickening of the right apical pleura; aortic sclerosis, slight degree.

Dorsal spine—hypertrophic changes, slight degree; less than usual for patient's age.

Lumbar spine—no abnormal features.

Sacral spine—spina bifida occulta of the first sacral segment.

Shoulder, right—(*a*) Irregular flattening of the articular surface of the head of the humerus. The borders are of usual density but there is a slight general subcalcification of the head, much more marked in the region of the greater tuberosity and in the upper half of the head. (*b*) The normal lamellar structures are destroyed in the humeral head in certain areas to the extent that there are two somewhat spherical areas totally devoid of lamellar structure; surrounding these, the bone is of irregular sclerotic density. (*c*) The joint space is of moderate width. In the lowermost portion of the joint are noted irregular calcified bodies, about 1.8 × 1.2 cm. in size. Similar bodies of smaller size are present in the uppermost areas of the articular cavity. These findings indicate chondromatosis of the joint.

Shoulder, left—changes very similar to those of the right, except slightly more marked; no changes from normal in the scapulæ.

Clavicle, right—two areas devoid of bone structure, about 1.2 and 1.4 cm. in size, of spherical shape, in the lateral fourth of the clavicle. The surrounding bone is somewhat coarser than usual but

does not present the sclerotic changes noted in other bones. Otherwise the cortex and medulla of the clavicles, as studied

thickening of the cortex on the medial aspect; upper portion of the neck is in juxtaposition with the upper and lateral

Fig. 4.

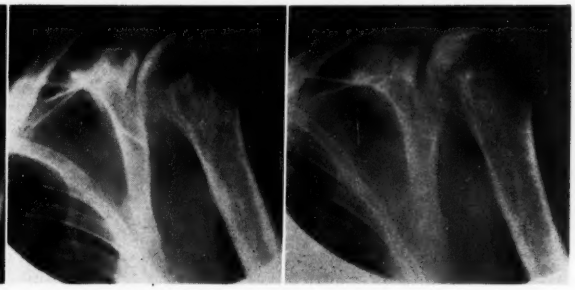

Fig. 5.

Fig. 4. Case 2. Necrosis in the femoral head. Secondary osteo-arthritic changes. Note similarity to findings in Case 1.
Fig. 5. Case 3. Sclerosis of subcartilaginous bone in each humeral head. Necrotic changes and medullary calcification.

from the x-ray standpoint, show no abnormality.

Long bones of the upper extremities—in the upper portion of the diaphysis of the humerus on each side, greater on the left, the lamellar structure in some areas is disturbed, but not as much as in the head. Extending through the medullary portion of the shaft distally from the head for about 17 cm. is a calcific body of irregular density and contour, varying in width from 0.5 to 1.5 cm., with well defined borders, and apparently due to previous necrosis. The cortex of the shaft is of normal thickness. There is no periosteal reaction.

Hands—a small chondroma is present in the proximal phalanx of the right thumb, of no apparent association with caisson disease. There is moderate deformity of the left fourth metacarpal, due to an old fracture.

Hip, right—(*a*) slight flattening and irregularity of the articular aspect of the femoral head; moderate homogeneous increased density of the upper half of the head; considerable coxa vara deformity due to depression of the head in relationship to the neck axis. (*b*) The femoral neck is broader than normal, with slight part of the acetabulum; this portion of the neck is bounded laterally by a protruding rounded area of its structure. The normal lamellation of the upper half of the head shows slight replacement by ovoid radiolucent areas not exceeding 0.5 cm. in size. (*c*) The articular space is markedly decreased in the upper half and slightly diminished in the lower part. The ilium in the upper and outer side of the acetabulum shows moderate increase in density, and flattening.

Hip, left—before operation, similar to the right, with symmetrical findings. After operation: (*a*) Evidence of an osteotomy; the femoral head is of irregular outline, wedge-shaped, and markedly denser than normal; slight coxa vara alignment. (*b*) The neck is displaced upward and somewhat lateral to the head. (*c*) The articular space is grossly irregular and decreased in its upper half. The contiguous portion of the ilium shows increase in density. The medial half of the hip joint is widened, due to the lateral shift of the femur.

Knees—similar. (*a*) The joint space is of normal depth and symmetry; the articular margins are smooth and regular; minimal hypertrophic changes of the tibial spines are present on the left. (*b*) There

is preservation of the normal lamellar architecture of the subcartilaginous bone; slight subcalcification.

periosteal reaction is present. There is slight subcortical atrophy of the left femur (side operated upon). These areas

Fig. 6. Case 3. Considerable medullary calcification in each femur and tibia. Normal joint surfaces.

Long bones of the lower extremities—in the distal portion of the femoral shafts and in the proximal areas of the tibial diaphyses are present irregular calcific changes in the medullary cavity similar to those in the humeri. The cortex is normal. No

of medullary necroses vary from 7 to 10 cm. in length, and from 0.5 to 3.5 cm. in width. They arise at least 1.5 cm. away from the epiphyseal zone, not invading it, and are broader nearer the joint.

Laboratory Data.—Jan. 22, 1937, uri-

nalysis, yellow, clear; specific gravity, 1.046; 1 plus albumin; 3 plus sugar; pus cells, 5 to 10 per low power field; occasional hyaline casts. Jan. 27, gonococcal fixation tests negative; blood Wassermann test negative. Jan. 29, blood chemistry:

urea, 46 mg. per 100 c.c. blood,
creatinine, 1.28 mg. per 100 c.c. blood,
sugar, 80 mg. per 100 c.c. blood,
uric acid, 3.15 mg. per 100 c.c. blood.

Feb. 11, spinal fluid: 67 mg. total protein per 100 c.c.; sugar, 53 mg. per 100 c.c.; no increase in pressure; fluid clear; negative globulin; red Benedict reaction; no increase in white blood cells; no organisms in smear; spinal Wassermann negative. Feb. 13 and 17, blood Wassermann negative. Feb. 23, blood count:

4,500,000 red blood cells,
7,800 white blood cells,
85 per cent hemoglobin,
32 per cent lymphocytes,
2 per cent eosinophils,
60 per cent polymorphonuclears,
6 per cent monocytes.

Feb. 24, blood chemistry:

urea, 43 mg. per 100 c.c. blood,
creatinine, 1.25 mg. per 100 c.c. blood,
sugar, 94 mg. per 100 c.c. blood.

Feb. 25, urinalysis: no sugar or acetone. March 11, biopsy (operation): the specimen consists of two pieces of bone, yellowish in color, very difficult to cut, and of firm consistency. Diagnosis: chronic hypertrophic osteo-arthritis.

Fig. 7.

Fig. 8.

Fig. 7. Case 3. Right hip normal. Osteo-arthritis of left hip with necrosis in the femoral head.
Fig. 8. Case 3. Necrosis in head of left femur. Moderate osteo-arthritis.

March 12, urinalysis: orange-yellow, cloudy, alkaline; specific gravity, 1.044; no albumin; no glucose; heavy sediment; amorphous and triple phosphates. March 18, April 9, April 23 to 30, sputum negative for tubercle bacilli (five times). Aug. 13, urinalysis: amber in color, clear; specific gravity, 1.026; negative for albumin and glucose; few pus cells. Feb. 8, 1939, blood Wassermann negative; gonococcal fixation test negative. April 12, 1939, blood chemistry:

calcium, 10.5 mg. per 100 c.c. of blood,
phosphorus, 2.79 mg.,
phosphatase, 6.2 units (Bodansky).

Temperature, pulse, and respiration were normal, except for a slight post-operative febrile episode of respiratory origin, in March, 1937.

Case 2. P. D., American white male, aged 55 years, admitted to the orthopedic service of Dr. Joseph B. L'Episcopo, Kings County Hospital, on May 24, 1939, with the diagnosis of caisson disease of the hips. The patient had worked as a "sandhog" for over 25 years, being employed on the construction of the Hudson Tube and other compressed-air work. He had had

several attacks of the "bends," the first of which occurred about 20 years before, followed by the onset of bilateral hip pain two years later. He has continued to do construction work under increased air pressure, with progressive pain in both hips. During the past three years he has walked with a marked waddling gait, and of late is unable to bend to tie his shoe laces.

Physical examination reveals marked limitation of flexion, extension, and abduction of both hips. There is slight limitation of flexion and extension at the lower lumbar level of the spine. There are advanced dental caries. Other findings are normal.

Laboratory Data.—Negative blood Wassermann and gonococcal fixation tests; sedimentation time, 120 minutes; temperature, pulse, and respiration normal. Blood chemistry:

calcium, 9	mg. per 100 c.c.,
phosphorus, 2.6	mg. per 100 c.c.,
phosphatase, 8	units (Bodansky),
urea, 23	mg. per 100 c.c.,
creatinine, 1.17	mg. per 100 c.c.,
sugar, 74	mg. per 100 c.c.

X-ray Examination.—A summary of the findings of the osseous system and the chest is as follows:

Fig. 9. Case 4. A survey of the osseous system revealed only two significant findings: medullary calcification in the lower part of the right femur, and a small area of increased density in the right ilium just above the hip.

Skull—no abnormal features.

Lungs—moderate calcified tuberculosis of both upper lobes, involving the apices and subapical areas; lower bronchi thickened.

Heart—slight enlargement of left ventricle and the aorta.

Dorsal spine—slight hypertrophic changes of the lowest vertebræ. Slight dextro-rotary scoliosis of the lower two-thirds.

Lumbar spine—slight hypertrophic lipping; slight dextro-rotary scoliosis.

Sacral area—no abnormal findings; calcified iliac vessels.

Shoulders—normal except for several small areas of radiolucency, about 2 mm. in size, in the head and neck of each humerus.

There is no medullary calcification of either humerus.

Long bones of the upper extremities—no abnormal features.

Hips—similar: (*a*) Flattening and irregularity of the articular surface of each

femoral head. There is a central area of radioparency with a moderate sclerotic border indicative of necrosis. Coxa vara deformity is present, due to medial depression of each head. (*b*) The femoral neck on each side presents no unusual changes. (*c*) The articular space is decreased throughout, with moderate sclerosis and hypertrophic changes of the upper half of the acetabulum.

Knees—no abnormal findings.

Long bones of the lower extremities—normal except for old healed fractures through the lower shaft of the right tibia and fibula.

There is no characteristic medullary calcification.

Case 3. W. M., born in Ireland, aged 67 years, was admitted to the orthopedic service of Dr. L'Episcopo, Kings County Hospital, on July 28, 1939, with the diagnosis of caisson disease of the left hip. The patient had been a "sand-hog" for 43 years, and claimed that he had suffered at least ten attacks of "the bends" while working under high pressure (47 pounds). He had been employed on all the tunnel construction work around New York City.

He related that he had been perfectly well until three years before, when his left hip began to feel "weak." This progressed until he is at present incapacitated for work. There are no other complaints except occasional slight pain in the left shoulder. Pain in the left hip is slight, although there is considerable disability, necessitating the use of a cane.

Physical Findings.—Slight impairment of hearing; moderate dental caries. No limitation of motion in the joints of the upper extremities; normal function of the right hip. The left hip shows moderate deficiency in flexion, extension, and rotation; no shortening; atrophy and weakness to a slight degree of all muscles about the hip.

A complete roentgen examination of the osseous system and the chest was made. A summary of the findings is as follows:

Chest—slight thickening of the bronchi; moderate sclerosis of the aorta; moderate enlargement of the left ventricle.

Shoulders—calcification in the medullary canal of the proximal portion of each humerus; moderate necrotic changes in each humeral head; also sclerosis of the subcartilaginous bone of each humeral head, greater on the right.

Elbows—no abnormal features except more than average productive changes of the olecranon process of the right ulna.

Wrists—no abnormal features.

Hips—right hip normal; moderate necrosis in the head of the left femur and moderate osteo-arthritis of the left hip.

Knees—no abnormal features of the joint surfaces; well defined medullary calcification in the distal portion of each femur and proximal portion of each tibia.

Ankles—no abnormal features except calcification of the vessels.

Spine—only slight hypertrophic changes.

Case 4. J. G., American, aged 44 years, was admitted to the surgical service, Kings County Hospital, on July 1, 1939, for treatment of fractures of the right leg.

He gave his occupation as a "sand-hog" and said that he had worked under compressed air (50 pounds) since 1921. He had had "the bends" three or four times since 1921. He has no joint disability but states that infrequently he feels moderate pain in each shoulder.

Physical findings were normal except for fractures of the right leg sustained in an automobile accident. Spinal tap showed a clear fluid, and a pressure of 3 mm. Hg.

A roentgen examination of the entire osseous system disclosed only a few positive findings. Except for moderate hypertrophic changes of the lateral end of the left clavicle and of the left elbow, of no apparent association with caisson disease, the only significant findings were:

Right hip—a small area of increased density is present in the lower part of the right ilium; the joint is normal.

Right knee—slight medullary calcification in the lower part of the right femur; the knee joint is normal.

Remarks.—This last case is interesting

because of the small amount of medullary calcification present and the absence of the osteo-arthritis found in the previous cases.

Another patient, W. B., aged 38 years, was admitted with a fracture of the right ankle on July 2, 1939. He gave his occupation as a "sand-hog," working on the Holland and other tunnels. He has never had "the bends." X-ray studies of the osseous system were negative for any evidence of caisson disease.

SUMMARY AND CONCLUSIONS

(1) Four cases of caisson disease are reported for record and to demonstrate certain roentgenographic features.

(2) The chronic bone changes in this disease include: (*a*) aseptic necrosis involving the hips, shoulders, or knees; (*b*) medullary calcification in the diaphyseal ends of long bones; (*c*) hypertrophic arthritis.

(3) Caisson disease may be manifested by aseptic necrosis with osteo-arthritis alone, as well as by medullary calcification.

The authors wish to express their appreciation to Dr. Joseph B. L'Episcopo, Director of Orthopedic Surgery at Kings County Hospital, for his kind co-operation in making the patients' records available.

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THE ROENTGEN-RAY EXAMINATION IN INDIVIDUALS SUFFERING FROM LOW BACK AND SCIATIC PAIN, WITH EXAMPLES OF SOME LESIONS IN WHICH ERRORS IN DIAGNOSIS MAY READILY BE MADE[1]

By JOSEPH C. BELL, M.D., *Louisville, Kentucky*

THE problem of low back and sciatic pain has been given much attention in medical literature during the past five years and, as a result, the medical profession in general is rapidly acquiring a better understanding of the abnormalities responsible for the symptoms and signs presented by persons suffering from these conditions. Increasing numbers of individuals are being referred to those presumably especially trained in the diagnosis of the many conditions which may be responsible for these symptoms.

Roentgenologists have made important contributions to the knowledge and understanding of these conditions and have, and in my opinion will continue to have, a sphere of major importance in this field of diagnosis. Recently in some centers there has been a tendency to resort to roentgenologic examinations only in those cases which do not present what are considered to be characteristic localizing signs and symptoms of intraspinal lesions. Neurologic localization in quite a high percentage of cases can be very accurate. Our recent experiences would seem to indicate, however, that the information obtained, and the errors avoided by a properly performed roentgen examination of the spina canal with some contrast medium, make this type of examination desirable in most cases in which operative intervention is under consideration. The operative approach now in use demands accurate localization. Formerly, wide exposures were employed, the dura opened, etc. Errors in localization of one segment, or at times more, usually did not preclude the discovery and removal of the offending lesions. In our recent cases, however, only a portion of the lamina over the suspected lesion has been removed, the dura and its contents retracted, and the cartilaginous mass, if found, removed. The dura has not been opened and no attempt has been made to remove the contrast medium.

Some of the illustrations forming a part of this communication show the findings in certain of our recent cases which seem to show the value of the roentgen examination. Others illustrate potential sources of error in diagnosis. Figures 1, 1-*A*, and 1-*B* are of an individual who had a severe spinal injury with a crushing fracture of the body of the twelfth dorsal vertebra more than a year prior to the time of our examination. She suffered from pain in the region of the fracture from the time of the injury and a few weeks later developed symptoms of low back disease. She was seen by Dr. R. G. Spurling who suspected an encroaching lesion in the spinal canal. He injected 2 c.c. of lipiodol into the lumbar subarachnoid space and referred her to the x-ray department of the Norton Memorial Infirmary for examination. Figure 1 shows serial exposures of the lower lumbar area of the spinal canal. An hourglass type of deformity is shown as the lipiodol column passed the region of the fourth lumbar interspace. As is routine in our department, even when obvious lesions are found low in the canal, the patient was turned on the table, the head lowered, and the progress of the column cephalward was observed. An almost complete block of the subarachnoid space was encountered at the level of the twelfth dorsal vertebra, as is shown in Figure 1-*A*. Figure 1-*B* shows the crushing fracture of the body of the twelfth dorsal vertebra previously mentioned. At operation post-traumatic

[1] Presented before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

adhesions were found at the level of the fracture. These apparently accounted for the marked encroachment on the subarachnoid space shown at our examination. The region of the fourth lumbar interspace was then explored and a large herniated nucleus pulposus was found and removed.

months progressive weakness of the lower extremities had been developing and had become so marked at times that she was unable to stand. The pain suggested a herniated nucleus but the progressive weakness was not in keeping with the usual manifestations of this condition. As is

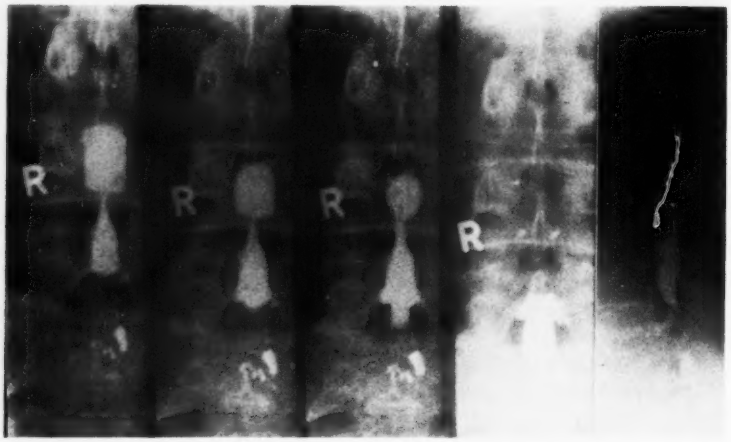

Fig. 1. Hourglass-like narrowing of canal at fourth lumbar interspace. Herniated nucleus found at operation at this level.

Figures 2 and 2-*A* are of a case quite similar to the one just described except that the second lumbar vertebra was fractured. This patient also continued to have pain in the region of the fracture after the injury and shortly thereafter developed low back pain which radiated into the left extremity. At operation post-traumatic adhesions were found in the region of the fracture together with a tear in the dura. Some of the nerve filaments were adherent to the overlying lamina which previously had been fractured. A herniated nucleus was found at the fifth lumbar interspace on the left and was removed.

Figure 3 shows exposures of the spinal canal at various levels in the case of an individual sent to the writer's office for examination. Through an oversight I had been given no information concerning the case. In brief, she told me that she had suffered from low back pain for more than a year, but added that for the previous eight

shown in the first exposure on the left in Figure 3, the root sleeve on the right at the level of the fifth lumbar interspace is absent. This could well be indicative of the presence of a herniated nucleus pulposus at this level. When the head was lowered, the column of opaque material passed cephalward with no delay until it reached the level of the eleventh dorsal interspace where a complete block of the subarachnoid space was encountered. The evidence of an obstruction is shown in the exposure on the extreme right in Figure 3. The two exposures in the center of the illustration show bizarre irregularities in the outline of the column, such as are sometimes seen in the lumbar area but more often observed in the dorsal region. Just why they occur I do not know. They may be due to intervertebral discs which protrude into the canal or to peculiarities in the development or position of the vertebral bodies themselves. When multiple, it is usually obvi-

ous that they are not significant from the clinical standpoint, but when single and unilateral, as in Figure 10, they may become a potential source of error in diagnosis. The volume of contrast medium has no apparent bearing on the occurrence of these defects for in this case 5 c.c. of lipiodol were used, but they also have been seen frequently when only 2 c.c. have been employed.

That the deformities sometimes seen in the lipiodol column may be caused both by peculiarities in the contour of the posterior aspect of the adjacent vertebral bodies as well as prominence of the intervertebral discs is suggested by the appearance of the lipiodol column in Figure 3-*A*. The illustration is not taken from the same examination as Figure 3, but is included since it seemed that the appearance of the column in the lateral view might throw light on the deformities seen in other individuals.

Figure 4 is of an individual thought, from the neurologic viewpoint, to have a lesion in the lower lumbar area. The x-ray examination showed no abnormality in the outline of the column in the lower lumbar region, but as it passed cephalward an almost complete block of the subarachnoid space was encountered at the level of the second lumbar interspace, as is shown in the two exposures on the right in Figure 4. Measurement of the spaces between the pedicles showed enlargement of the canal at the level of the first and second lumbar vertebræ. Operation revealed a large hemangioma of the conus.

In Figure 5 a large hourglass defect in the column is shown at the fourth lumbar interspace. In addition, however, the third exposure from the left shows a small defect at the fifth lumbar interspace on the left, with elevation of the adjacent root sleeve. Operation revealed a herniated nucleus pulposus impinging upon the root of the fifth lumbar nerve on the left. It had displaced the nerve upward and there were adhesions between the nerve and the nucleus. The adhesions were separated by blunt dissection and the nucleus removed. A markedly hypertrophied ligamentum flavum was found at the level of the fourth interspace but a careful exploration failed to reveal any evidence of a herniated nucleus at this level.

Figure 6 is very similar in appearance to Figure 5. An hourglass constriction was present at the level of the fourth inter-

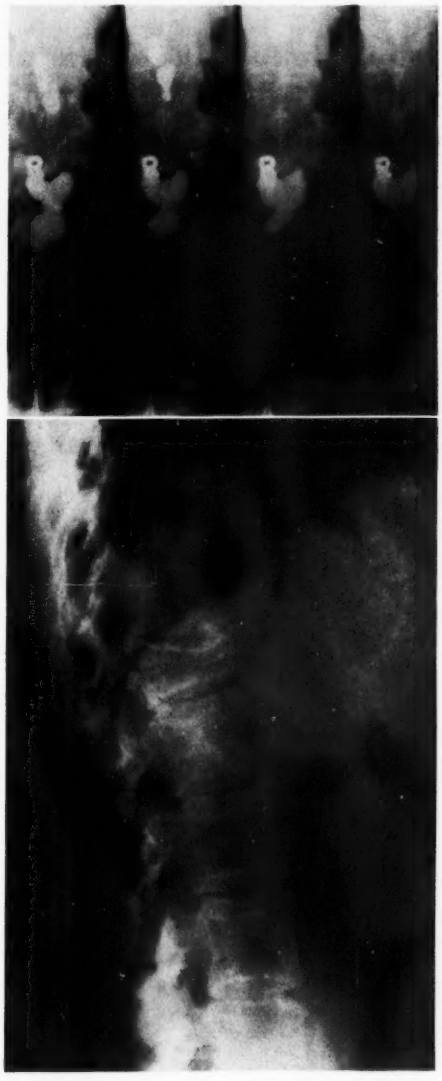

Fig. 1-*A* (*above*). Partial block of subarachnoid space at level of twelfth dorsal vertebra following fracture of the body of this vertebra.

Fig. 1-*B* (*below*). Lateral view showing fractured twelfth dorsal vertebra in same patient presenting the abnormalities shown in Figures 1 and 1-*A*.

Fig. 2 (*above*). Anteroposterior exposures showing partial obstruction of subarachnoid space at the level of the second lumbar vertebra following a fracture of this vertebra.

space, and the root sleeve on the left at the level of the fifth interspace was elevated. At operation a herniated nucleus was found at the fourth interspace with some of the extruded material in such a position that it had made pressure on the intradural portion of the left fifth lumbar nerve and caused the displacement noted. There was no evidence of a herniated nucleus at the fifth lumbar interspace.

Figure 7 shows a large filling defect on the left at the fourth lumbar interspace and a relatively small defect on the same side at the fifth lumbar interspace. A markedly hypertrophied ligamentum flavum was found at operation at the fourth interspace and a herniated nucleus at the fifth.

Figure 8 shows an elevated root sleeve on the left at the level of the fifth lumbar interspace. It shows how slight may be the changes which, if constant and if supported by a thorough neurologic examination, will justify a diagnosis of a herniated nucleus and its exact location. The lateral view on the right showed no defect. This exposure was made with the fluoroscope without the use of the Bucky diaphragm. At operation a herniated nucleus was found impinging upon the root of the corresponding nerve as it passed into the intervertebral foramen.

Figure 9 also shows how slight may be the changes justifying a diagnosis of a herniated nucleus if supported by the neurologic findings. The two exposures on the left show the column of opaque material to be displaced to the left at the level of the fifth lumbar interspace, and show a very small curved defect on the right at this level. The root sleeve of the nerve on the left may be seen but not the one on the right. The lateral film showed the lumbosacral joint space to be narrowed and the column of lipiodol to lie unusually far posterior to the posterior aspect of the body of

Fig. 2-*A* (*below*). Lateral view of lumbar spine of same patient as shown in Figure 2, showing fractured second lumbar vertebra and also ventral defect in lipiodol column at fifth lumbar interspace due to herniated nucleus.

the fifth lumbar vertebra, suggesting the presence of a mass within the canal encroaching upon the ventral aspect of the space at this level. At operation a large herniated nucleus was found at the level of the fifth interspace on the right.

Figure 10 is a series of exposures showing the column of lipiodol passing from the sacral sac upward to the level of the second lumbar vertebra and then downward into the sacral sac again. A defect is shown on the left at the level of the fourth interspace. It was best demonstrated when the column was relatively thin and almost disappeared when the column was thick, as was the case in the second exposure from the right. A second defect is shown on the right at the level of the third interspace. It was ignored because there was no neurologic evidence of an encroaching lesion at this level. At operation a herniated nucleus was found on the left at the level of the fourth interspace. (See p. 456.)

In the two illustrations on the left in Figure 11, lipiodol is shown in the subdural space. This may be confusing for the column will move to a certain extent, but not freely, and in some ways will resemble the appearance seen in an individual who has an arachnoiditis. In this case the neurologic surgeon insisted that the lipiodol must be in the subarachnoid space for he had withdrawn fluid both before and after injection. It seemed evident from the roentgenologic standpoint, however, that such was not the case and a second injection was done, with the results shown in the three exposures on the right. The two exposures made in the anteroposterior position show the root sleeve on the left at the

Fig. 3 (*above*). Exposure on left shows absent root sleeve on right at fifth lumbar interspace consistent with herniated nucleus. The second and third exposures show the peculiar defects in the column of contrast material that are sometimes encountered. These had no apparent clinical significance. The last exposure on the right shows a block of the subarachnoid space at the level of the eleventh dorsal vertebra shown at operation to be caused by a meningioma. Pedicles were not eroded. Lower lumbar area was not explored.

Fig. 3-*A* (*below*). Lateral view of lumbar spine and pelvis showing column of contrast medium at the level of the third lumbar vertebra. A small curved filling defect is seen in the ventral aspect of the column at the third lumbar interspace. The relationship of the structures here suggests that this defect may be due both to prominence of the adjacent vertebral margins and to prominence of the intervertebral disc. There was no neurologic evidence of an encroaching lesion at this level. The patient had herniated nucleus on right at fifth lumbar interspace which was removed.

fifth lumbar interspace to be markedly elevated, while the lateral view shows two columns of opaque material, one presumably being in the subdural space and the other in the subarachnoid space. At operation a nucleus was found on the left at the level of the fifth interspace and was removed. The dura was opened and quite a large amount of oil was found in the subdural space. (See p. 456.)

Figure 12 shows a large defect in the lipiodol column on the left at the level of the fourth lumbar interspace. The tip of the sacral sac is at the lower margin of the fifth lumbar interspace. This is an example of a sacral sac of the type described by Camp, who called attention to cases in which the lower extremity of the canal did not extend into the sacrum and said that herniated nuclei at the fifth lum-

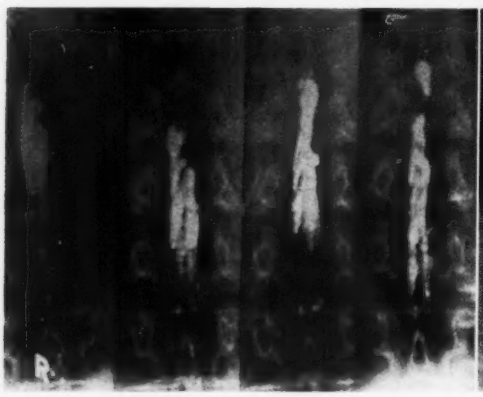

Fig. 4.

Fig. 5.

Fig. 4. Partial obstruction of the canal at the level of the second lumbar vertebra, due to hemangioma of conus. The canal was somewhat dilated at this level.

Fig. 5. Hourglass-like deformity of the column at fourth lumbar interspace, due to markedly hypertrophied ligamentum flavum. Small defect and elevated root sleeve on the left at fifth lumbar interspace due to herniated nucleus.

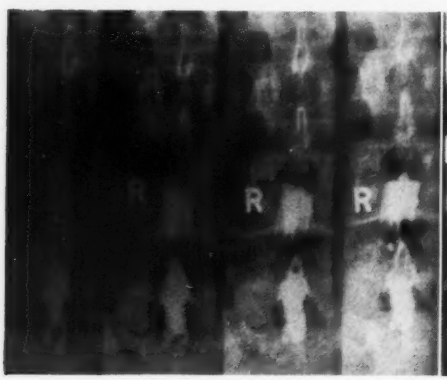

Fig. 6.

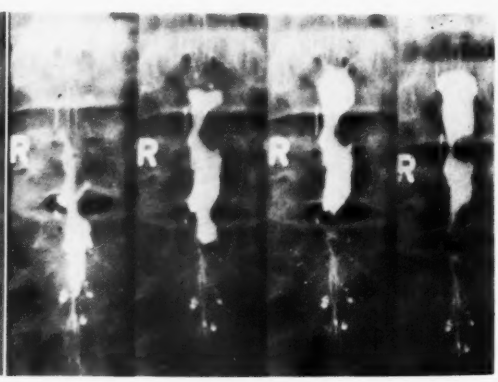

Fig. 7.

Fig. 6. Hourglass-like deformity of column at fourth lumbar interspace due to large herniated nucleus. This nucleus also caused the elevation of the root sleeve on the left at the fifth interspace shown in the illustration.

Fig. 7. Deformity of canal on left at fourth lumbar interspace due to hypertrophy of ligamentum flavum. Small curved defect with absent root sleeve on left at fifth lumbar interspace, due to herniated nucleus pulposus.

bar interspace could seldom be diagnosed by x-ray in cases such as these. At operation a herniated nucleus was found on the left at the fourth lumbar interspace and a second herniated nucleus was found on the same side at the fifth lumbar interspace. They were removed. The lesion at the fourth interspace is obvious in the illustration but no evidence of a lesion at the fifth interspace can be seen. (See p. 457.)

general physical examination, and a neurologic examination by one experienced in the diagnosis of the various abnormalities that may cause low back pain. The injection of a contrast medium into the spinal canal should not be resorted to unless, in the opinion of the above mentioned examiner, definite evidence of an encroaching intraspinal lesion exists.

The contrast material used in the examination is a matter of personal preference. We use lipiodol, the amount injected being 2 c.c. No undesirable sequelæ which could be attributed to this

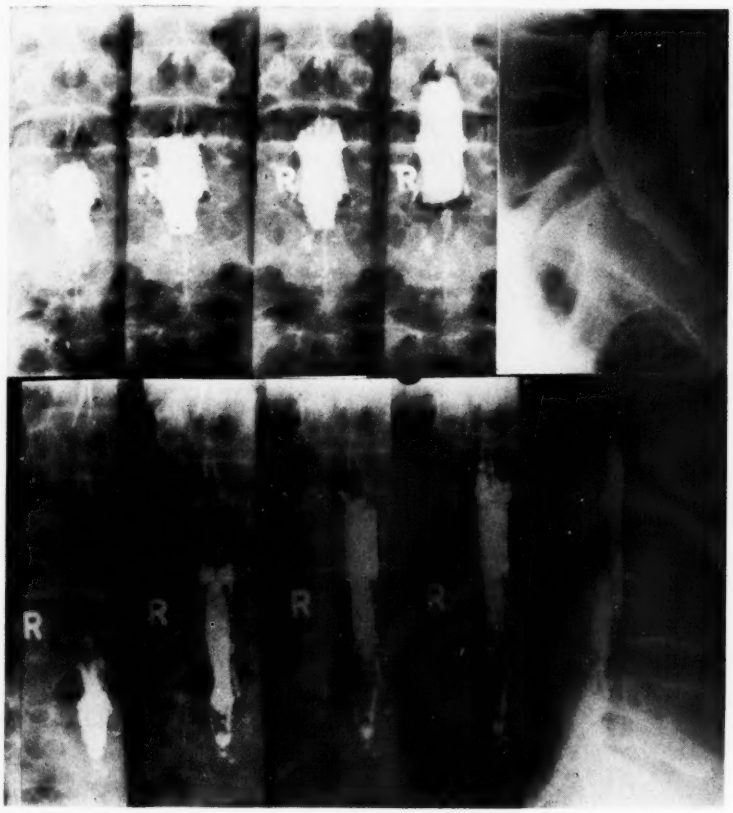

Fig. 8 (*above*). Slight elevation of root sleeve on left at fifth lumbar interspace due to herniated nucleus. Lateral view made with fluoroscope without a grid. It shows no irregularity in the outline of the column.

Fig. 9 (*below*). Slight curved defect on the right and slight displacement of column of contrast material to left at fifth lumbar interspace due to herniated nucleus. Lateral view shows, rather unsatisfactorily, narrowing of lumbosacral joint space and ventral filling defect in the column at this level.

Comment.—Accuracy of diagnosis in the field of low back pain demands that certain requirements be met. One essential is that the patient have a complete history, a

substance have been observed in any of our cases. The amount injected is also a matter of personal preference but in our hands 2 c.c. have seemed entirely adequate. Lipiodol casts a dense, sharply defined shadow and one in which minor defects may readily be detected. Such minor defects may be of even more importance from the clinical point of view than many gross ones. Our experience with air as a contrast medium is inadequate but it seems inconceivable that small defects such as are shown in some of the foregoing illustrations can be demonstrated by this medium.

We are firmly convinced of the value of an adequate number of films of satisfactory quality made with the column of contrast medium passing over each interspace in the region of suspected pathology. In my work I seldom make less than eight exposures in the anteroposterior position with the patient prone and usually at least two additional exposures in one of the lateral positions. One group of four exposures is usually made as the column passes down toward the sacral sac and the other as it progresses cephalward. When a defect persists in a series of exposures of

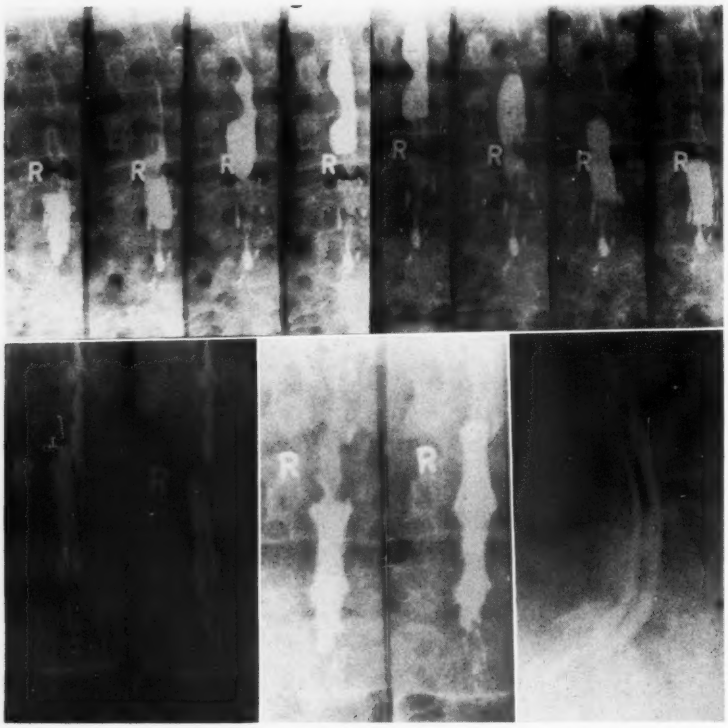

Fig. 10 (*above*). Serial exposures made as column of lipiodol passed upward and downward through the lower lumbar canal. Small filling defect is shown on the left at the fourth lumbar interspace, proven at operation to be due to a herniated nucleus. A relatively large filling defect is shown at the third lumbar interspace on the right but there was no evidence of an encroaching lesion at this point and the area was not explored. The defect was not of the type usually seen in the presence of a herniated nucleus but might be due to unilateral hypertrophy of the ligamentum flavum.

Fig. 11 (*below*). The two exposures on the left show contrast medium in subdural space. The third and fourth exposures show lipiodol in the subarachnoid space. Root sleeve of the nerve on left at the fifth lumbar interspace is elevated, due to herniated nucleus. Last exposure on right shows lateral view of contrast material in both the subdural and subarachnoid spaces.

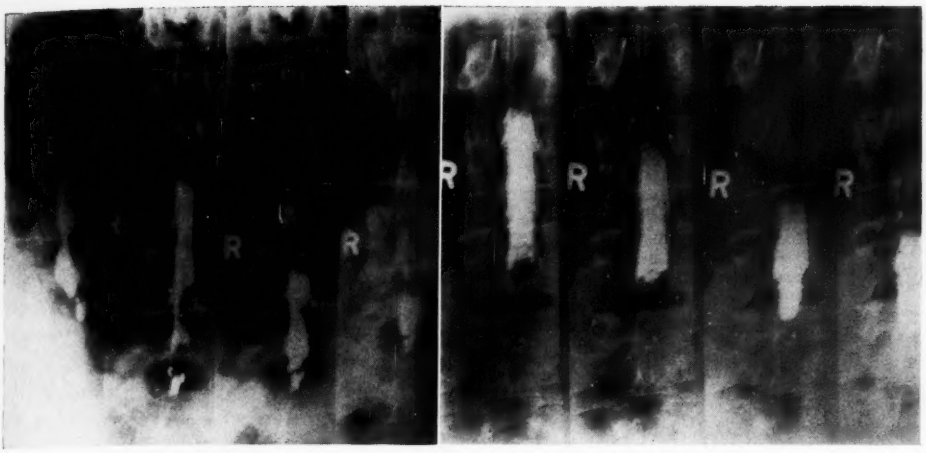

Fig. 12. Fig. 13.

Fig. 12. Curved defect on left at fourth lumbar interspace, due to herniated nucleus. Second nucleus found on left at fifth lumbar interspace but not shown, due to the fact that the sacral sac extended downward only to the superior margin of the sacrum.

Fig. 13. Contrast material in lower spinal canal. Root sleeve on the left just above fifth lumbar interspace is definitely elevated. Herniated nucleus at this point found at operation and removed. Patient had previously had studies of lower spinal canal, air being used as a contrast medium. The films were examined in two institutions and no evidence of any abnormality was found.

this type, one may be quite certain that it has an organic basis.

When a lesion in the lower lumbar area is suspected, serial films of other parts of the canal are not made unless a defect in the column or a block is encountered. In all cases, however, regardless of defects which have been demonstrated low in the canal, the column is carefully observed until it passes high into the dorsal area.

In some instances we have been unable to distinguish between defects caused by hypertrophied ligamenta and those caused by herniated nuclei. This can readily be appreciated from a study of some of the illustrations forming a part of this paper. From the clinical standpoint, however, such differentiation has not seemed to be of major importance.

A herniated nucleus is accompanied by an hypertrophied ligamentum flavum at the same level in a large percentage of cases. Marked hypertrophy of a ligamentum flavum may be present at one level, however, and a herniated nucleus at another. Early in our work ignorance of this fact was responsible for some errors in

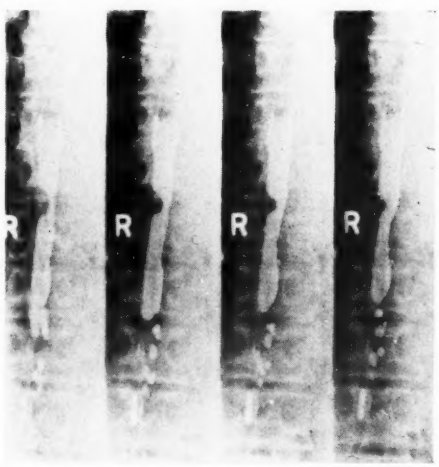

Fig. 14. Serial films of upper dorsal portion of spinal canal showing complete block of subarachnoid space at level of fourth dorsal interspace. At operation the block was shown to be due to an extradural mass which was encroaching upon the ventral aspect of the canal. The mass apparently was an extruded portion of the fourth dorsal intervertebral disc. A small filling defect can be seen on the right at the level of the sixth dorsal interspace. Operation showed no evidence of a herniated nucleus at this level. The defect probably was due to unilateral hypertrophy of the corresponding ligamentum flavum. We are not certain of this, for the ligament was not examined carefully at the time of operation.

diagnosis, in that the defects due to hypertrophied ligaments were observed and minor ones due to herniated nuclei were overlooked.

Hypertrophy of the ligamentum flavum is unquestionably a definite entity, but associated pathology is probably responsible for the major symptoms presented in most cases. In cases in which an hypertrophied ligament causes gross narrowing of the canal, however, it would seem unwise not to remove it even when other pathology is found which could explain most or all of the patient's symptoms.

The findings in the two traumatic cases presented in the illustrations suggest that it might be wise to resort more often to a study of the spinal canal with some contrast material, in those patients who suffer severe spinal trauma and who do not have a satisfactory convalescence. This is especially true in those who suffer fractures of the vertebral bodies or posterior arches. In the two cases mentioned, each suffered pain at the site of fracture from the time of injury but developed symptoms of low back disease some weeks later. The herniation of the nuclei seemed definitely to be associated with the injuries.

To my mind, the most important safeguard to error in this field of diagnosis is the closest possible coördination of the neurologic and roentgen-ray findings. The neurologic findings, however, are the cardinal determining factors in evaluating the importance of defects demonstrated and in indicating whether or not operative intervention is advisable.

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MYELOGRAPHY WITH THE USE OF THORIUM DIOXIDE SOLUTION (THOROTRAST) AS A CONTRAST MEDIUM[1]

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From the Cleveland Clinic

As with many another substance which may eventually prove to be harmful if used indiscriminately, the status of thorium dioxide solution (thorotrast) in the literature appears to be determined largely by each author's inclination to approve or disapprove of its use. Either contention may be supported by experimental evidence based on the results of the administration of comparatively large doses in both animals and humans. This substance, in small amounts, probably possesses no greater potentialities for harmful effects in the body than many other diagnostic and therapeutic substances now in use. If given in carefully calculated doses which are below the minimal estimated toxic level, we feel that it may be possible to take advantage of some of its valuable properties.

In the report of the Council on Pharmacy and Chemistry (1), the properties of thorium dioxide solution (thorotrast) are outlined. Its composition, as given by the manufacturer, is said to be a stabilized thorium dioxide solution containing 25 per cent by volume of thorium dioxide (from 19 to 20 per cent by weight) and from 16 to 19 per cent by weight of dextrin preparation as a protective colloid. There is added to the solution 0.15 per cent methyl-*p*-hydroxybenzoate as a preservative. It is felt by those who have employed it that, excepting exploratory surgery, it yields diagnostic information not obtainable by other means. The problem of ultimate toxicity is the main consideration, since the immediate reaction to its use is slight.

The apprehension regarding the use of thorotrast is based primarily upon its radio-active properties. Thorium and its preparations disintegrate into mesothorium, radiothorium, thorium X, thorium emanation, thorium A, B, C, C′, C″ (2, 3), until the products of disintegration accumulate and reach equilibrium with each other (4). Despite the low penetrating powers of the alpha ray, this ray is believed by Flinn (5) to be of greatest importance in its effects upon tissue, the ratio of toxicity for the alpha, beta, and gamma rays being given by Martland (6) as 10,000 to 100 to 1, respectively, while their relative penetrations vary in the reverse order.

The radio-activity of thorotrast has been summarized in the report of the Council as follows:

"Alpha ray—Twenty-five c.c. of thorotrast is equivalent to a maximum of one microgram and a minimum of 0.5 microgram of radium (Schlundt).

"Beta rays—'Too feeble to be of physiological significance' (Schlundt).

"Gamma rays—Twenty-five c.c. of thorotrast is equivalent to 0.4 microgram of radium (Radium Institute Bergakademie, Freiberg); equivalent to approximately 0.3 microgram of radium (Flinn). 'Too feeble to be of physiological significance' (Schlundt)."

The significance of these figures will be considered later but it is sufficient to state that in this study they afford the basis for calculating the radio-activity of the retained thorotrast. Further, it is important to note that whereas formerly the limit of tolerance for radio-active substances was thought to be equivalent to 10 micrograms of radium in the normal person, the incidence of radio-activity intoxications makes evident the possibility that 1 or 2 micrograms may produce toxic alterations (Flinn, 5).

[1] Presented before the Twenty-fifth Annual Meeting of the Radiological Society of North America at Atlanta, Dec. 11–15, 1939.

Among the toxic effects, various authors list as attributable presumably to various radio-active substances are the production of severe leukopenia with acute nephritis and purpuras (7, 8) by the alpha ray, peritoneal and subcutaneous sarcomas by thorium dioxide (9), osteogenic sarcoma (10, 11), and aplastic anemia.

It is the factor of fixation of the thorium dioxide in the tissues, particularly through the agency of the reticulo-endothelial system, which has presented a major problem, although this factor in all probability makes possible hepatosplenography, osteomyelography, pneumo-alveolography, and placentography. It is to be borne in mind that unless some mechanical method for removal is employed, the effective radioactivity probably can be calculated from the amount of thorium solution injected, since very little, if any, elimination of this material takes place. Irwin (12) believes that a small portion of this material may be transported intracellularly from the liver to the lungs and eliminated in the bronchial mucus, although most investigators are inclined to believe that no significant elimination occurs (13, 14, 15, 16).

Fig. 1. Myelogram made with colloidal thorium dioxide, showing the presence of a herniation of the intervertebral disc of the lumbosacral joint. Verified by operation. (Postero-anterior and lateral views.)

In encephalography, ventriculography, and myelography, the problem is somewhat altered by the fact that the thorium dioxide is injected into the cerebrospinal fluid

which is removable by suitable methods. It is in this phase of subarachnoidal roentgenography that our interest is particularly centered.

The use of thorium dioxide solution injections into the subarachnoidal spaces is not new. Radovici and Meller (17, 18), of France, and Wustmann (19), and Löhr and Jacobi (20, 21), of Germany, employed it experimentally in 1932. They quickly appreciated the clarity with which the nervous structures could be demonstrated but their enthusiasm was somewhat dampened by the serious reactions and even deaths which followed its injection. The problem of retention in the tissues and the precipitation of the colloid by a rise in the acidity of the cerebrospinal fluid often constituted a reaction to its use. After considerable experimentation, thorotrast, an alkaline, buffered suspension of thorium dioxide in a protective colloid dextrin, made its appearance and, while a slight acidosis in the subarachnoid space may still occur, the material in this form is well tolerated.

In 1936, Freeman, Schoenfeld, and Moore (22, 23) and later, Twining and Rowbotham (24) recommended the use of thorotrast in ventriculography because its great radio-opacity, its miscibility with the cerebrospinal fluid, and high specific gravity result in the production of fewer reactions than usually attend air ventriculography. It is the feeling of Reeves and Stuck (25), as well as others who have experimented with this material, that long retention of colloidal thorium dioxide on the surfaces of the central nervous system produces an aseptic meningeal reaction with resultant phagocytosis of the material. However, this reaction is not unlike that attendant upon the injection of any foreign substance into the subarachnoid space.

If it were possible to utilize in myelog-

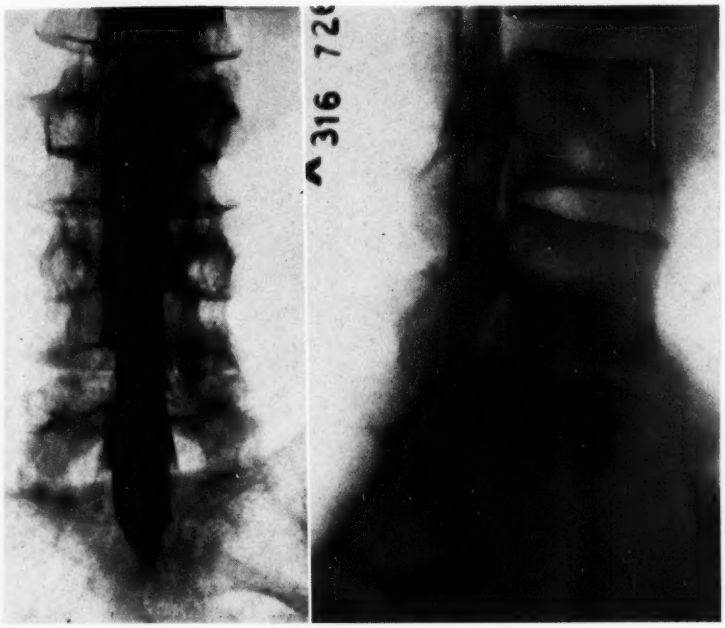

Fig. 2. Myelogram made with colloidal thorium dioxide, showing herniated nucleus pulposus between the fourth and fifth lumbar vertebræ. Verified by operation. (Postero-anterior and lateral views.)

raphy the favorable elements of thorotrast, namely, its heavy atomic weight and high specific gravity and its diffusibility in the cerebrospinal fluid, and then eliminate it from the subarachnoid space after it had served its purpose in such a roentgenographic study, it would represent a valuable addition to the neurologic armamentarium. It occurred to one of us (Nosik) that, because of its miscibility with cerebrospinal fluid, much of a subarachnoidal thorotrast injection could possibly be removed by the forced cerebrospinal fluid drainage method of Kubie and Retan (26). A series of experiments with dogs and monkeys was done by Nosik and Mortensen (27) to test the practicability of such a procedure. It became apparent at once that the clinical reactions of the animals not subjected to a forced cerebrospinal fluid drainage after thorium dioxide injection were quite severe although not as disastrous as those reported by Coe, Otell, and Hedley (28) or those experienced by Spurling (29) in his trial of the material. In the undrained animals, definite signs of an irritative meningeal reaction persisted for several days, while those animals which had been submitted to a forced drainage on completion of the thorotrast myelograms were free from such reactions. The clarity of the subarachnoidal detail and the

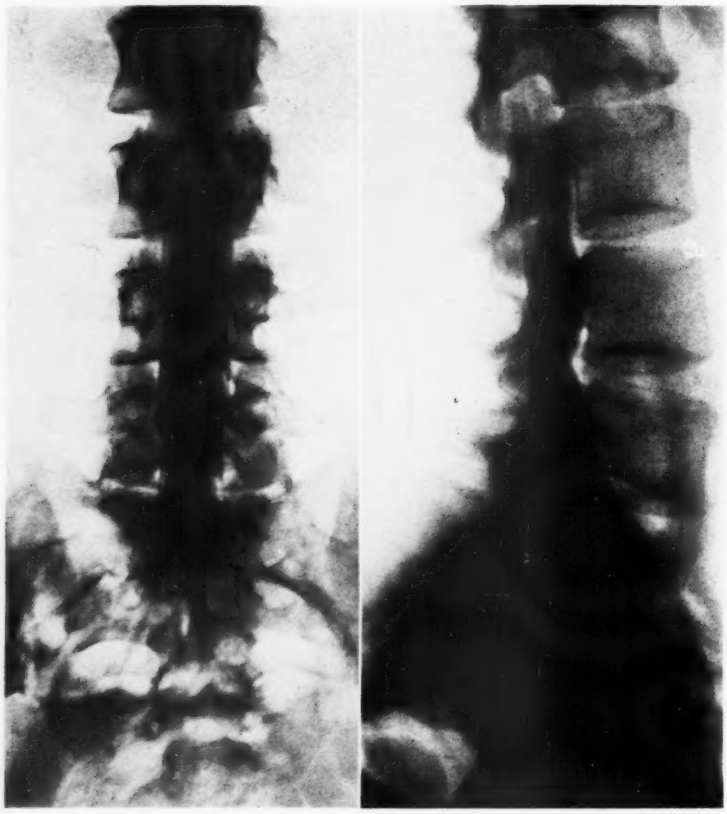

Fig. 3. *A*, Myelogram made with colloidal thorium dioxide, showing herniated ruptured intervertebral disc between the fifth lumbar and first sacral vertebræ. Verified by operation. *B*, Myelogram demonstrating herniated intervertebral disc between the fourth and fifth lumbar vertebræ in lateral view.

completeness of the removal of the thorotrast usually obtainable by this technic made it obvious that in the experimental animal, at least, the theoretical considerations were established in practicable form.

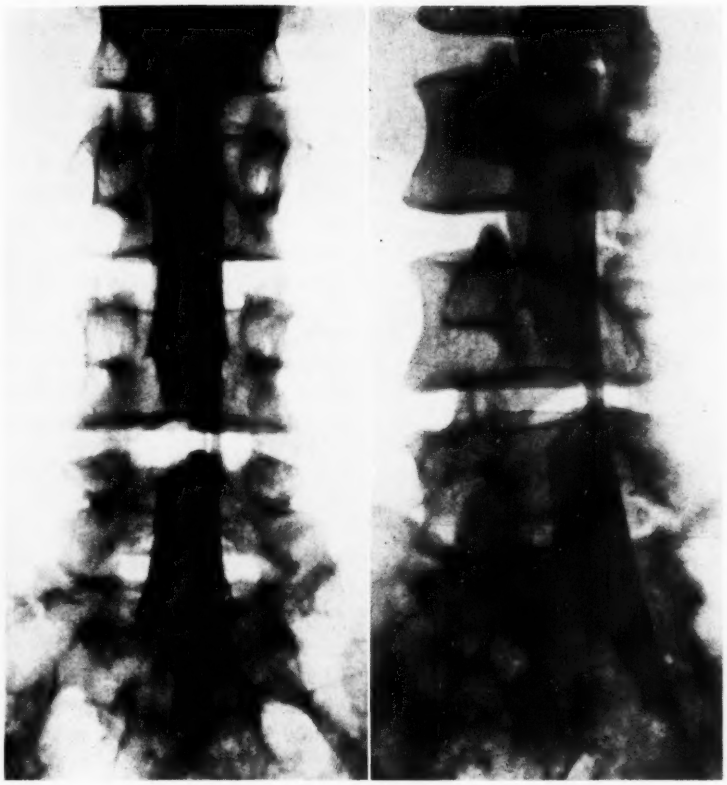

Fig. 4. Myelogram made with colloidal thorium dioxide, showing a ruptured nucleus pulposus with herniation between the fourth and fifth lumbar vertebræ. Verified by operation. (Postero-anterior and lateral views.)

After becoming better acquainted with the difficulties usually attendant upon any new enterprise and being more firmly convinced of the practicability of this technic of myelography, attention was next directed to its clinical application. The reactions of the undrained animals were so much like those of the reported human cases (none of which had been drained thoroughly) that it was felt a forced cerebrospinal fluid drainage would be equally effective in the elimination of both the thorium dioxide and the reaction in the human subject. Accordingly, the first trials of this technic of myelography were done by Nosik (30). Injections of predetermined amounts of thorotrast into the lumbar sac were made and roentgenograms taken. Upon completion of the roentgenography the patients were subjected to a forced cerebrospinal drainage of the thorotrast-containing cerebrospinal fluid. This fluid, together with a typical 1 c.c. specimen of the thorotrast used in the injection, was submitted for a quantitative analysis to determine the percentage return of the thorium dioxide. It was apparent from the films that this method of myelography could have a definite place in the roentgen

diagnosis of partial space-filling lesions of the spinal canal. It was also found that by forced drainage the reactions to this material were markedly lessened and the thorium dioxide was eliminated to a greater degree than any other radiopaque substance used in the diagnosis of these lesions up to this time.

In the myelograms presented herein (Figs. 1, 2, 3, 4, and 5), the amount of thorotrast injected into the canal varied from 6 to 10 c.c., satisfactory myelograms being obtained even with the smaller amounts. The quantitative recovery of the thorium dioxide injected ranged from 70 to 90 per cent. To consider a typical case of this series in which 8 c.c. of thorotrast was injected and a recovery of 80 per cent was made: Using the radium equivalent of thorotrast as accepted by the Council, it is apparent that the total maximum alpha radiation present in the injected thorotrast is equivalent to 0.32 microgram of radium. However, of this injected amount, 0.256 microgram of radium equivalent is recovered; hence it remains that in this case an alpha radiation equivalent to 0.064 microgram radium is retained in the body. Likewise, to consider the radiation of this amount of thorotrast, using the maximum figures of gamma ray contained in thorotrast as given by the Council, it is found that the injected amount of thorotrast contains 0.1280 microgram radium equivalent of the gamma ray. However, of this amount a recovery of 0.1024 microgram radium equivalent was made; hence the gamma radiation remaining in the body is equivalent to that of 0.0256 microgram radium. To recall the figures previously given, changes in the bones and teeth were found in the bodies of radium workers containing from 2 to 4 micrograms of radium, while an amount as low as 1 microgram of radium is said to have given evidence of focal atrophy and sclerosis in the mandible in some radium dial workers. This seems to be the minimal toxic amount in which changes have been observed. Obviously, the figures for our average case reveal that the maximum radiation from the retained thorotrast is far below the minimal reported toxic amounts, giving this substance a margin of safety far greater than that possessed by many another substance used diagnostically and therapeutically.

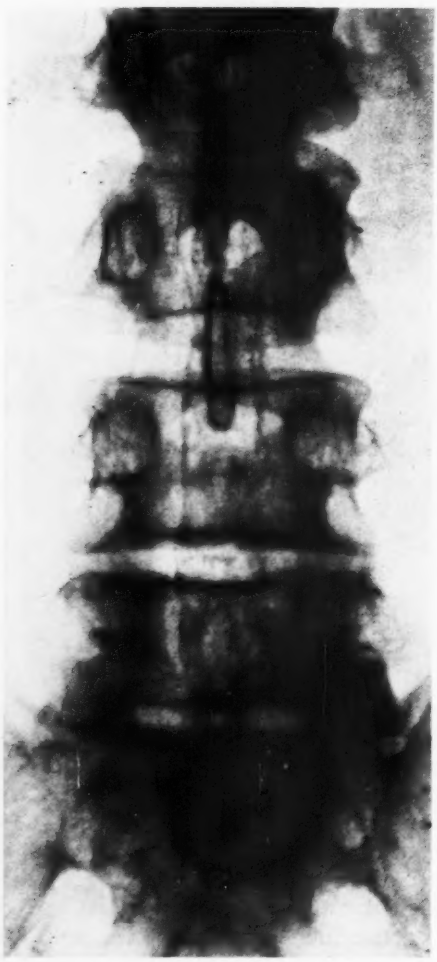

Fig. 5. Film taken after removal of colloidal thorium dioxide by forced cerebrospinal fluid drainage.

This technic for thorotrast myelography represents a combination of experimentally and clinically proven procedures, *i.e.*, visualization of bodily structures by thorotrast and the forced spinal drainage of Kubie and Retan. The search for an easily visualizable agent, which by its mis-

cibility with the cerebrospinal fluid will readily outline the nooks and crevices of the subarachnoid space without masking a lesion and which can be eliminated after it has served that purpose, is not over. But, to overcome some of the inherently undesirable qualities of the most suitable contrast medium of this type for cerebrospinal visualography yet developed, namely, thorotrast, a technic for its elimination was applied here, followed through its animal application to test its practicability, and, finally, applied successfully to the human. It is simple enough to be done by those familiar with the principles involved, and the authors feel that with more experience in this method, highly satisfactory myelograms can be made.

From the nature of the procedure it is evident that a subarachnoidal block is a contra-indication to this method of examination. It should be reserved for partially obstructive or non-obstructive lesions in the canal—the very lesions which present the greatest diagnostic problem and the most favorable surgical possibilities. It is felt that the utilization of thorotrast in the technic described is an improvement in clinical myelography. The application of forced cerebrospinal fluid drainage and the removal of diagnostic substances from the patent canal is fundamental, when permitted by the dictates of their physical properties.

CONCLUSIONS

1. Myelography with thorotrast and its removal by a subsequent forced cerebrospinal fluid drainage has been applied clinically and found to be a practicable and useful procedure.

2. The application of forced cerebrospinal fluid drainage to the removal of a diagnostic substance of suitable physical properties from the patent subarachnoidal space is presented here and is considered to be of basic importance.

3. The recovery of a high percentage of the injected colloidal thorium dioxide is possible by this method.

4. The residual thorium dioxide in the bodies of these patients represents a radium equivalent of alpha and gamma radiation far below the minimal reported toxic level.

5. The clinical reaction to this procedure is much like that of an ordinary forced cerebrospinal drainage.

The authors wish to express their appreciation to Dr. W. James Gardner and Dr. James A. Dickson for their co-operation in the compilation of this manuscript.

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THE EFFECT OF IODIZED OIL ON THE MENINGES OF THE SPINAL CORD AND BRAIN[1]

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THE use of contrast media is a well established and necessary adjunct to the roentgen examination of many parts of the body. It is desirable that such media should be safe, reliable, simple in application, and readily available. For general purposes, substances of high contrast are preferable, especially in the examination of thick or dense parts of the body. Substances of high atomic weight such as thorium, iodine, bromine, and their derivatives or compounds are commonly used. Substances of low contrast, such as dilute preparations of the above named elements and, various gases are often unsatisfactory because slight variations in their thickness (indicating the presence of organic lesions) may easily be overlooked. In the investigation of diseases of the spinal cord and spinal canal, one of the safest and most satisfactory opaque media yet developed is iodized poppy seed oil (lipiodol), containing 54 per cent iodine in combination. By the use of this medium, Camp (4), Robinson (19), and many others (9, 10) have shown that diseases of the spinal cord and associated tissues can frequently be discovered when all other diagnostic methods, including pneumomyelography, have failed. Such an opaque medium is, therefore, of great value and, if safe, its use should be approved, at least until a superior one has been developed.

Since iodized poppy seed oil remains in the cerebrospinal system once it has been injected into the subarachnoid space, its mere presence has been held to blame for innumerable symptoms which patients with organic disease of the spinal column or spinal cord have developed subsequent to its injection (6, 15, 16). Well known physicians have stated that, "the use of lipiodol in outlining the lumbocaudal sac and in locating the level of a complete block is unjustified" (25). "Due to the *likelihood* of bad effects, we have attempted to substitute air for lipiodol" (26). "Of all the complications (of spinal canal lesions) the worst is the presence of iodized oil as shown by the x-ray films" (25). In view of these astounding, and in our opinion erroneous, statements, it is believed that the clinical and pathologic reactions attributed to lipiodol should be reviewed, and the status of its alleged harmful effects brought up to date.

EXPERIMENTAL OBSERVATIONS

Experimental work on animals was reported by Ayer and Mixter (1) in 1924. Using large doses of iodized sesame oil (in amounts approximately five times as great as conventional amounts used in humans), they found high spinal fluid cell counts following cisternal injection.

Davis, Haven, and Stone (6), in 1930, reported the results of intracisternal injection of lipiodol in 10 dogs. In eight of them they found a severe leptomeningeal reaction and concluded that the injection of iodized oil into the subarachnoid space was a dangerous procedure. They did not report the findings in control dogs. All of their animals had artificially produced subarachnoid blocks.

In 1931, Lindblom (12) performed a critical investigation on the effects of various iodized oils on the meninges. He found that the degree of irritation caused by the oil varied with the amount of free fatty acid: sesame oil with an acidity of

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0.6 produced no irritation; refined poppy seed oil with an acidity of 2.4 produced moderate irritation; while ordinary poppy seed oil with an acidity of 6.5 produced marked irritation. He has found that the oil was found to have collected in large lakes outside the cerebral hemispheres while in the other, it was distributed in numerous small droplets, especially in the basal cisternæ. When the brain was re-

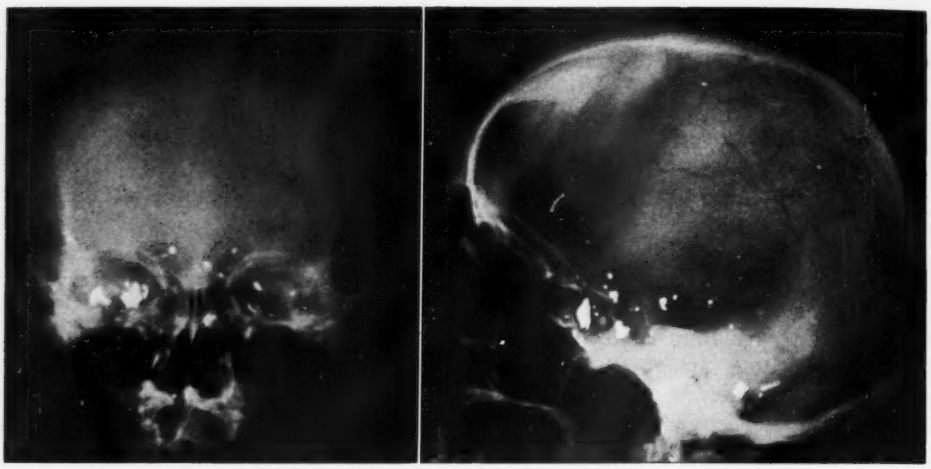

Fig. 1. Roentgenogram of the skull of N. S., a laborer, who had 10 c.c. of lipiodol injected into the cisternal subarachnoid space *14 years* previously! This patient, then aged 58, had symptoms of a spinal cord tumor. The large amount of lipiodol was used in error (from 3 to 5 c.c. being the usual amount). No roentgen evidence of tumor was found and the patient was not operated upon. He improved gradually and now hobbles around with the aid of two walking sticks. He has large amounts of lipiodol scattered throughout the skull, chiefly in the basal cisternæ and about the cerebellum, and a large lake of about 5 c.c. in the spinal *cul de sac*, still movable! He has no symptoms or neurologic findings attributable to the oil.

the iodized product of the oil caused greater irritation than the oil itself, the free fatty acid being transformed into iodic fatty acid. He regarded lipiodol as an impure iodized oil but found that its impurities were absorbed comparatively easily, while the iodized oil itself was absorbed slowly. He noted in rabbits that after the injection of oils that were absorbed slowly, a sort of "pseudotumor" or fatty granuloma was sometimes found in the subarachnoid cavity. These granulomas did not always develop and when they did develop they took at least six months. Two rabbits were given injections of 1 c.c. each of lipiodol and were killed after six months, Before the brains were extracted the skulls were roentgenographed. In one, moved from the former, two large lakes of oil were found on it, surrounded and infiltrated with new-grown connective tissue. At another place there was a pachymeningitic reaction, which on incision also showed droplets of oil. Histologic examination showed that these formations consisted of fatty phagocytes of various sizes, surrounded or infiltrated with new-grown connective tissue (fatty granulomas). In the other specimen, in which the oil was separated into small droplets, there were no granulomas; numerous small droplets were found in the arachnoid membrane itself, but without reaction. Lindblom observed, "The literature contains a few cases of dissection on humans after lipiodol injection; judging from the histologic de-

scriptions it seems likely that fatty granulomas were present in some of them. These granulomas appear to develop when the oil gathers in large lakes. Further investigation is required to confirm this point and *also to determine whether or not these granulomas have any clinical significance.*"

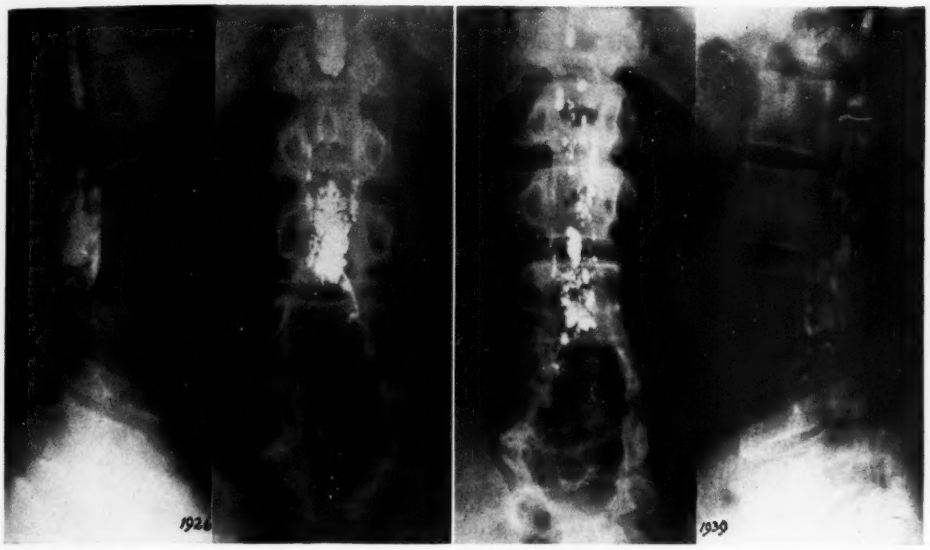

Fig. 2. Roentgenograms of the spine of F. B., an elevator operator, who had 5 c.c. of lipiodol injected into the cisternal subarachnoid space *14 years* previously! This patient, then aged 27, had symptoms of spinal cord tumor. Roentgen examination disclosed marked erosion of the dorsal aspects of the lower lumbar bodies, and complete block of the opaque oil at the level of the third lumbar disc. Laminectomy disclosed an "inoperable neurofibroma," only a small portion of which could be removed. The patient gradually improved and is still working. He has no detectable symptoms resulting from the lipiodol. Large droplets of oil remain in the spinal canal; there are a few droplets in the basal cisternæ and cerebral ventricles. Most of these are fixed. The roentgenograms on the left were made in 1926, those on the right in 1939.

Bruskin and Propper (3) reported the results of experimental myelo-encephalography in dogs in 1931. They injected iodipin subdurally and found no disturbances of sensation or motor function. Pathologic examination after from one to three months revealed granulation-tissue formations over the spinal cord; in some areas there was destruction of the gray substance of the cord itself; however, it should be noted that the oil was injected *into* the cord itself in two dogs. These authors believe that the iodine and oil split up, the former producing the reaction.

CLINICAL AND PATHOLOGIC OBSERVATIONS

Immediately after the subarachnoid injection of fresh lipiodol in amounts of from 2 to 5 c.c., there is an increase in the white cell count of the spinal fluid which lasts for about four days. There is sometimes mild hyperemia about the site of injection. In some patients there are symptoms of mild headache, cramps in the lower limbs, and mild accentuation of the backache of which the patient has so often been complaining. There is sometimes a transitory rise in temperature of about 2 degrees Fahrenheit. This reaction lasts for one or two days and usually subsides without leaving any residual symptoms. Sicard and Forestier (22, 23), who introduced lipiodol for diagnosis as well as therapy, stated that in patients whose meninges were inflamed or congested as a result of recent trauma, "contact of the oil with the

diseased area sometimes caused a transient but quite endurable pain." They noted that in paralytics there was sometimes an increase of spasticity and slight inhibition of the sphincters. In tabetics there were short crises of shooting pains, tingling of the lower limbs, and transitory disturbances of the sphincters. In all of their cases reactions of this kind disappeared completely within four days. They believed that a simple lumbar puncture was capable of producing similar effects in certain cases. They noted that the opaque oil disappeared very slowly from the cerebrospinal system.

Fig. 3. Roentgenograms of the skull of H. W., a physician's wife, who had 3 c.c. of lipiodol injected into the lumbar subarachnoid space almost *3 years* previously! This patient, aged 30, had backache and right-sided sciatica for two years. Myelography disclosed a right-sided defect at the level of the fourth lumbar disc. At operation pieces of displaced disc and nuclear material were removed along "with as much oil as possible." At the present time there are small lakes of opaque oil in the lumbar area, a few droplets elsewhere in the spinal canal, and the above numerous droplets and globules in the skull. These are in the cisterna pontis and posterior fossa, and are fixed. The lumbar collections are movable. This patient has no neurologic findings or clinical symptoms referable to her cranial cavity, and no symptoms attributable to the oil in the spinal canal.

Maclaire (14), in 1925, and Sharpe and Peterson (21), in 1926, reported what they believed was a case of severe meningeal inflammation following the use of lipiodol. Their patient had sustained a fractured fourth thoracic vertebra; a few years later signs of spinal cord compression and irritation developed. Subarachnoid injection of lipiodol revealed a block at the level of the seventh thoracic vertebra. Laminectomy was performed at the level of the fourth thoracic vertebra; nothing abnormal was found and the incision was closed. The patient grew worse and five months later laminectomy was performed at the level of the tenth thoracic vertebra; some lipiodol was found encysted in adhesions and there was considerable local arachnoiditis. Maclaire and Sharpe believed that the lipiodol alone brought on the meningeal inflammation. Sicard, discussing this case, believes that the adhesions and arachnoiditis antedated the injection of the oil, but again emphasized the occasional disturbances produced by lipiodol in cases of old spinal traumatism. Ebaugh and Mella, in 1926, reported a

transient aseptic meningitis following intracisternal injection of iodized oil in 13 cases. Wolfsohn and Morrissey, in 1927, reported inflammatory changes around tumors in two cases following lipiodol injection.

arachnoid space and showed no evidence of any adhesive process." In one instance a necropsy was performed three and one-half months after injection; they found no evidence of any inflammatory process of the meninges at the site at which the

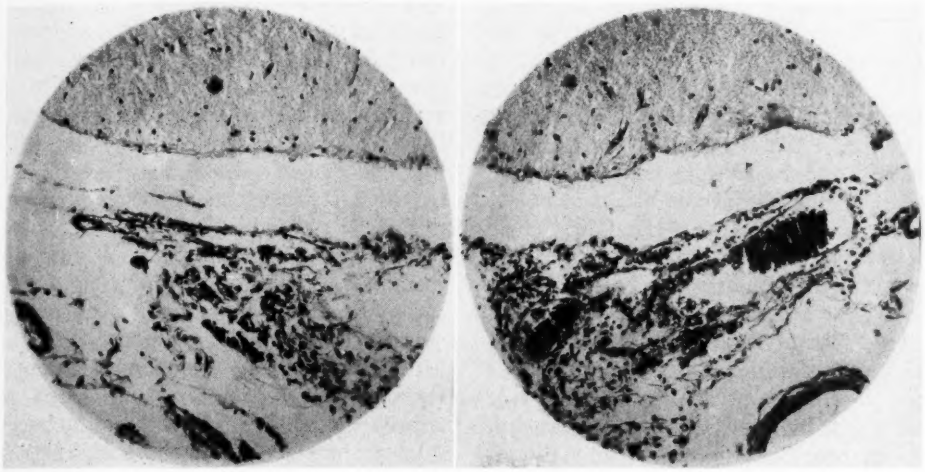

Fig. 4. Photomicrographs from two of several small nodules (pseudogranulomas) found at autopsy on the cerebellum of a laborer who had 3 c.c. of lipiodol injected into the lumbar subarachnoid space one year previously. This patient, aged 40, died of multiple sclerosis and chronic glomerulonephritis. The lipiodol had been injected to confirm or exclude the presence of a spinal cord tumor at the level of the seventh thoracic segment. No tumor was found. He had multiple, peculiar clinical symptoms and neurologic findings both prior and subsequent to the injection of the oil. When, at autopsy, numerous small yellow granules were found scattered over the meninges of the brain and cord it was at first thought that he had severe leptomeningeal and possibly neurologic changes from the lipiodol. However, histologic studies revealed no lesions in the neural tissues proper, in the vicinity of the lipiodol; merely small areas of fibrous proliferation, round-cell infiltration, and a few foreign body giant cells (containing minute fat deposits) in the *arachnoid* itself. The lipiodol droplets lay chiefly on the meninges over the lower 5 cm. of the spinal cord and the cauda equina, and over the base of the brain.

In 1929, Globus and Strauss (9) reported the results of subarachnoid injection of lipiodol in 64 cases. They concluded that in only one out of this group was there any clinical evidence of meningeal irritation; in this patient there was transient vasomotor disturbance and pain in segments approximating the level of the oil and tumor. They noted that 25 patients who had had iodized oil in their spinal canals for more than two years were normal upon examination, both roentgenologically and neurologically. Although the oil showed little diminution in bulk, "it remained freely movable within the sub-

iodized oil had been arrested. They believe that the iodized oil was absorbed extremely slowly but that it remained freely movable. Elsberg, in discussing this paper, stated, "I thoroughly believe that iodized oil is an irritant. I have seen in a number of instances fresh adhesions and marked congestion of the meninges, changes which are not ordinarily observed when the spinal cord is exposed during laminectomy; I have always explained this as the result of the irritative qualities of this foreign substance."

In 1932, Fumarola and Enderle (7) reported one alleged fatality following the

cisternal injection of lipiodol, but gave no histologic details. Hampton and Robinson, in 1936, reported the absence of any serious or permanent reaction following the use of 2 c.c. of lipiodol in over 100 examinations, and 5 c.c. in 75 examinations. They observed that the oil often produced a definite reaction, lasting for several days, characterized by elevation of the spinal fluid cell count, changes in the color and composition of the fluid, headaches, increase in the pain of which the patient complained, and slight fever. They comment that those who use the oil most frequently find the fewest reactions. They state that the oil did not become encysted even after two years. However, they do not recommend the indiscriminate use of the oil "and examine the upper thoracic spine only when symptoms referable to that portion of the canal are present . . . which may account for the infrequency of some of the untoward reactions which others attribute to the use of lipiodol." At the time of their report they did not seem to realize that the oil travelled up to the upper spinal regions and even the skull in a majority of cases, when the patient returned to bed or resumed his occupation.

The use of iodized oil in the study of the subarachnoid spaces of the skull and the cerebral ventricles was first attempted in 1926. In 1932, Sicard and Forestier advised that its use for this purpose be discontinued owing to early reactions in the form of severe headache and pyrexia. However, they reported that no untoward late effects were noted in their cases. These authors also reported on the use of "ascending lipiodol," a special light oil containing only 11 per cent iodine, in the examination of the ventricles; they tried it both plain and emulsified with spinal fluid. They reported that the latter mixture was extremely irritating and advised against its use in ventriculography.

In 1933, Brandt (2) reported the case of a patient with tuberculous spondylitis in whom 2 c.c. of iodipin had been injected into the lumbar subarachnoid space five and one-half months prior to autopsy. At autopsy numerous bright yellow nodules, varying from 1 to 4 mm. in diameter and resembling grapes in shape, were seen about the base of the brain, being especially numerous in the region of the basal cisternæ. These appeared to lie between the pia mater and the brain. They were easily movable and burst on slight pressure. Chemical examination showed that these nodules contained iodine. Microscopic examination showed only a structureless membrane. *No pathologic changes attributable to the iodine could be found in the neighboring brain.*

Ouchi (18), in 1935, reported 25 cases in which he had used opaque oil for myelography. In several of these cases he noted mild early reactions, such as nausea, irritability, pyrexia, and so forth, all of which disappeared within a week. In one instance he erroneously injected the oil in a case of tuberculous meningitis; shortly thereafter the patient died. Ouchi attributed the patient's death to the meningitis and not to the lipiodol. However, he believed that it may well have aggravated the patient's disease. In seven cases he believed the oil exercised a therapeutically beneficial influence. In no cases did he meet with late harmful effects nor did he think the oil showed any tendency to become encapsulated or form granulomas, "as described in the literature."

Schüller (20), in 1936, investigated patients with possible tumors at the base of the brain by suboccipital or intraspinal injection of 40 per cent iodipin. He recommended this contrast filling of the basal cisternæ because: (1) the material was stable and there were no immediate or late unfavorable reactions; (2) the material was easily visible under the fluoroscopic screen, and (3) following investigation of the basal cisternæ the opaque oil could be displaced into the spinal subarachnoid space and thereby retained for further, later investigations of the cisternæ. His article contains nine illustrations showing oil in the skull, mostly in the form of lakes.

Lysholm (13), in 1938, discussed his experience with ventriculography, using

both air and opaque oils, based on a total material of 3,408 cases during 10 years. Of these cases, 123 were lipiodol examinations. Concerning the latter he comments that undesirable reactions sometimes occurred and that air is preferable and much less harmful.

Garland and Morrissey (8), in 1940, reported on a group of 25 cases, 23 of whom had had lumbar and two cisternal injections of lipiodol several months or years prior to examination. In most cases 5 c.c. had been used, but in one case, in error, 10 c.c. had been injected. This particular case was a patient with a fibroma of the lumbar portion of the spinal canal; he has been followed now for 14 years and has no symptoms attributable to the opaque oil, all of which is still in the cerebrospinal system. Of the entire 25 cases, approximately two-thirds showed collections of opaque oil intracranially, and all showed varying amounts in the spinal portion of the cerebrospinal system. All of the patients were examined neurologically and their symptoms and findings compared with those on their records prior to the time of injection of the opaque oil. In none of these cases were there any symptoms or neurologic findings which were not present prior to the injection or which could be ascribed to the presence of the oil.

SUMMARY OF CLINICAL AND PATHOLOGIC REACTIONS

Following the injection of small amounts (from 2 to 5 c.c.) of fresh lipiodol into the lumbar subarachnoid area there is often slight pain about the sacrum and coccyx, and sometimes mild headache and fever.[2] These symptoms usually subside spontaneously within five days. In a small percentage of cases, coccygeal pain is relatively severe, and this may persist for as long as three months, finally subsiding spontaneously.

The immediate pathologic changes consist of increased spinal fluid cell count and slight hyperemia about the site of injection of the oil. The late pathologic changes consist of small, round-cell and fibroblastic proliferation about the oil, especially about the smaller droplets which become scattered along the spinal canal. These small droplets often become encapsulated, resulting in miliary nodules on the surface of the cord (pseudogranulomas). In about two-thirds of cases, some oil is present in and about the basal cisternæ of the skull, usually in droplets which gradually become encapsulated.

The major quantity of the oil tends to persist in one or two fairly large "lakes," which remain movable for an indefinite period, and may be used for exploring the spinal canal months or years after the original injection.

We have previously (8) reviewed the autopsy findings reported in the literature, including the detailed findings in one case in which the opaque oil had lain in the cerebrospinal system for approximately one year. It has been emphasized before and should be repeated here that many of the tiny droplets plus their surrounding fibrous proliferations resemble miliary tubercles or granulomas of any type, and at first glance have led pathologists to conclude that some serious lesion of the brain or cord was present. Histologic examination will reveal that the changes are confined to the meninges and that the underlying neural tissues are normal (at least as far as any secondary effect from the lipiodol is concerned).

SUMMARY

Fresh iodized poppy seed oil (lipiodol) may be injected with safety into the subarachnoid areas of the spinal canal. The main drawback to its use is that it remains visible indefinitely in roentgenograms of the patient's cerebrospinal system. Such roentgenograms frequently cause apprehension on the part of the patient and his physician, to say nothing of such compensation boards and juries as happen to see them. However, opaque

[2] Similar symptoms are not infrequently noted after simple spinal puncture alone, as well as after spinal puncture and air injection.

materials such as mercury and bismuth have remained in patients' tissues for years and are visible in roentgenograms of their pelves and other areas. They have remained in these tissues without causing any harm; indeed, like lipiodol, they have been used both diagnostically and therapeutically with benefit. Until a superior opaque contrast medium has been discovered, we believe that the use of lipiodol is thoroughly justified *in cases in which it is indicated*. Its indiscriminate use, or its use without complete preliminary clinical, laboratory, and roentgenologic study remains unjustified—as does the use of any other contrast medium, gaseous or liquid.

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DISCUSSION

John D. Camp, M.D. (Rochester, Minn.): I was very much pleased with the way Dr. Bell presented his material. I think that he emphasized what is quite an important part of this whole problem. We all understand the various fundamental defects associated with the roentgenologic recognition of protruded intervertebral discs, but he emphasized that we must be sure, as the result of our examination, to rule out the presence of associated pathology which may be lurking in the background and masquerading as a protruded disc.

Our experience certainly has borne that out. We have a group of cases, as I have

said on previous occasions, of low thoracic tumors which have imitated discs neurologically and of course it would be a tragedy to take out a disc and leave a tumor in such a patient.

Statistics indicate that many patients with tumors in the spinal canal may have low back or sciatic pain. I do not mean that the tumor always produces the pain, but in our series 50 per cent of the patients with tumors in the thoracic area of the spinal cord have complained of low back or sciatic pain and 30 per cent of our patients with tumors in the cervical canal have complained of low back and sciatic pain. This emphasizes, I think, the necessity of a thorough neurologic examination before any contrast agent is introduced into the spinal subarachnoid space.

I think the amount of iodized oil one cares to use is probably not so extremely important, if we confine our observations to the lumbar region of the spinal canal. Dr. Bell elects to use 2 c.c. I prefer to use 5 c.c. I agree with Dr. Bell that 2 c.c. will probably show the majority of protruded intervertebral discs in the lumbar area. However, I think that whenever we put iodized oil into the spinal canal, the examination is not complete unless we rule out pathology above the conus, and from my experience I feel that I am unable to rule out efficiently small, non-obstructing tumors without 5 c.c. of oil. Smaller amounts of oil will pass by a small tumor without revealing its presence.

As to the differentiation between the deformity which results from the presence of a hypertrophied ligamentum flavum and that of a protruded intervertebral disc, I think it is an academic distinction. We know that practically all protruded discs have an associated hypertrophy of the ligamentum flavum, so the roentgenologic distinction is not important; it is just a little nicety in diagnosis which is sometimes gratifying to make. I feel that many times one can make a diagnosis of hypertrophy of the ligamentum flavum with or without the presence of a disc.

I have had no experience whatever with thorotrast. I would like to compliment Dr. Nichols on this beautiful piece of research. From his roentgenograms and exhibit, there is no doubt that thorotrast has some advantages over iodized oil so far as radiographic possibilities are concerned. Thorotrast shows the nerve roots beautifully and would be an ideal means to point out some of these small tumors. Unfortunately, for the present at least, the use of thorotrast seems confined to visualization of the lumbar canal. My only hesitancy in wanting to use thorotrast or recommending it would be that it is an intense irritant when it is in the soft tissues and I know that it is impossible to do lumbar punctures, at least many of them, without getting some of the medium in the epidural space occasionally.

I have seen only one patient in which a study of the lumbar subarachnoid space by means of thorotrast was attempted. Fortunately, the injection was not done at our institution. Following the injection, the patient promptly developed paralysis of the bladder. Roentgenograms at the time of his admission to the Clinic showed a large amount of thorotrast in the lumbar canal and all the nerve roots were well outlined.

Dr. Garland mentioned one point which I would like to emphasize; that is, the necessity of using fresh lipiodol. Fresh lipiodol is transparent and has a very, very faint yellow color. When lipiodol begins to deteriorate with the liberation of free iodine, it is an intense irritant. I think many of the so-called reactions from lipiodol are due to the use of deteriorated oil. You should be very careful in selecting an ampoule of lipiodol to be sure that it does not have a brown tinge to it. If it has, it should be discarded.

I do not know whether or not it is permissible for a discussant to discuss remarks of a previous discussant but I was a little intrigued by Dr. Fincher's[1] remark that air was preferable to oil. I like air

[1] Dr. Fincher's discussion not returned for publication.

very much. I happen to like lipiodol a little better, and my reason for liking lipiodol is based on the fact that in my experience lipiodol has been more accurate than air for this work.

I think there is a very definite place for air, or oxygen myelography. We have made about five hundred examinations with it and will continue to use it. However, I think that when one uses air, he should use it advisedly, and with a full appreciation of its diagnostic limitations and accuracy.

In our experience, a positive air study is worth about 83 per cent. However, in the cases in which we made a negative diagnosis from the air films, we were wrong in 77 per cent of the patients who were operated upon. Maybe there has been something wrong in the way I have examined these films. I certainly have looked at them hard enough! Nevertheless, so far as we are concerned, a negative air study is not worth very much. It helps us to rule out a space-occupying lesion in the lumbar canal fairly well but as far as determining the presence or absence of a protruded disc, I do not believe a negative spinogram is worth much at present.

You might summarize the situation by saying that if you get a positive finding with air, you are that much ahead. If the findings are negative, then you must take stock of the situation and decide whether you are going to use iodized oil or thorotrast or whether you want to be guided by the patient's symptoms and explore him directly.

X-RAY THERAPY OF HYPERPARATHYROIDISM[1]

By E. A. MERRITT, M.D., *and* RALPH M. CAULK, M.D.,[2] Warwick Memorial Clinic and Garfield Memorial Hospital, *Washington, D. C.*

THE relationship between the parathyroid glands and osteitis fibrosa cystica is well recognized. This subject was thoroughly presented by Merritt and Lattman (1) at the Annual Meeting of this Society in Detroit, in 1935, together with a review of the literature and case reports.

Shelling (2), in his admirable treatise, "Parathyroids in Health and in Disease," classifies hyperparathyroidism into three clinical types: (*a*) classical form in which the lesions of osteitis fibrosa cystica predominate; (*b*) osteoporotic form in which the decalcification of the skeleton is generalized and no tendency toward cyst or giant-cell formation is present, and (*c*) metastatic form in which the chief symptoms are associated with calcium deposition in the soft tissues, especially the kidneys, where it occurs in the form of calcinosis or of calculi.

The fundamental pathologic process in hyperparathyroidism is osteoporosis. Quoting from Shelling (2):

> "It differs from senile osteoporosis and from atrophy due to disuse in that in the latter instances the thinned compact and spongy bone is rarely replaced by fibrous tissue, whereas in hyperparathyroidism the resorptive and reparative processes are characterized by the presence of fibrous tissue in place of the original bone and marrow spaces; by the abundance of osteoclasts and by the presence of resorbed and newly formed bone. Some of the areas of fibrous connective tissue contain immature bone, others newly formed bone which merges with the original bone, and still others show no evidence whatsoever of new bone formation. In the fibrous areas which are free of bone tissue and which are often present subperiosteally, multinucleated giant cells and extravasated blood may be seen."

The aforementioned pathologic process occurring in the vertebral column produces a decalcification and collapse of the bodies, as a result of which the intervertebral discs expand into the cancellous bone, producing the so-called "fish-tail" spine. The end-result is a marked kyphosis, scoliosis, or both, associated with shortening of the stature and deformity of the thoracic cage which, in turn, alters the normal relationship of both the thoracic and abdominal viscera.

This presentation will be limited to a discussion of the spinal osteoporotic type and the symptom-complex resulting therefrom.

Clinically this form occurs most frequently in women past the menopausal age. The chief complaint is intense pain in the back, which is constant, is aggravated by motion, and often becomes worse when the patient is in the reclining position. Associated with this is a profound asthenia and some loss in weight. These symptoms have usually existed over a period of several years. Associated with these are often vague gastric and cardiac symptoms resulting from the disturbed visceral relationships produced by the kyphosis, scoliosis, and vertebral collapse.

The blood picture is that of a mild secondary anemia. Blood calcium and phosphorus determinations have been so unreliable in our experience that we do not consider these tests absolutely necessary to arrive at a diagnosis.

The roentgenographic picture of the spine is that which we would expect from the pathologic process described above, *viz.*, marked demineralization of the bone with collapse of the bodies and the typical "fish-tail" appearance of the spine. Kyphosis and scoliosis are present in varying degrees, depending upon the extent and duration of the disease. Multiple myeloma, metastatic carcinoma, and basophilic pi-

[1] Presented before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

[2] From the radiological clinic of Drs. Groover, Christie, and Merritt.

tuitary adenoma may give a similar picture and must be differentiated if possible. We feel that the therapeutic test of irradiation over the parathyroid glands is sufficient to rule out or confirm the presence of the disease in doubtful cases.

These cases do not present symptoms referable to the other joints or long bones. Demineralization of the remaining bones, other than the spine, may be present, but one can readily see that a critical analysis of this process, unless of marked degree, is very difficult to evaluate roentgenographically.

Since 1932, we have treated 14 such cases in our clinic. The technic has varied somewhat but at present the following is the general procedure: the factors are 220 kv., 20 ma., 50 cm. focal–skin distance, 2 mm. copper filtration, and a portal 8 × 15 cm. over the neck, anteriorly, reaching from the hyoid bone to the upper mediastinum. The first series consists of a total dose of 1,000 r (measured in air) given in from five to ten days' time. It is often necessary to repeat this series once or twice at six-week intervals. Beginning improvement in the symptoms may be anticipated from the tenth to fourteenth day and is usually progressive. The administration of calcium and vitamins as an adjunct to treatment has been of little or no value.

Recalcification is extremely slow, if it occurs at all. The existing deformities persist as we would expect, and as such are compatible with a relatively normal existence

Following is a report of one of our recent cases.

White female, aged 65, admitted to Garfield Hospital complaining of pain in the lower back. Family history and past history were essentially negative. Onset of the present illness was four months prior to admission, beginning with pain in the small of the back, which was sharp and stabbing in character. This pain did not radiate. At the time of admission the patient had a constant dull ache in the lower portion of the back but the stabbing character of the pain was intermittent. There was a gradual increase in the severity of the pain prior to admission, at which time it was excruciating. History of weakness could not be elicited, due to the predominance of pain. The patient had been treated by a chiropractor with no relief. She had lost 16 pounds in weight in the last year. General physical examination was essentially negative. Gastrointestinal examination was done and found to be negative. X-ray examination of the spine in the lateral projection revealed deformity of the articular surfaces with dorsal kyphosis, lipping of the vertebral bodies, narrowing and biconcavity, with extreme decalcification. The patient's height was 59.5 inches, weight 133 pounds.

Laboratory Findings.—At the time of admission serum calcium was 14.6 mg. per 100 c.c., serum phosphorus, 2.1 mg. per 100 c.c.; Wassermann and Kahn tests negative; urine negative; hemoglobin, 62 per cent; red blood cells, 3,270,000; white blood cells, 8,250; polymorphonuclears, 62 per cent, and lymphocytes, 37 per cent. The patient was given treatment over the neck, anteriorly, with 220 kv., 50 cm. distance, 10 × 12 portal, Thoraeus filter equivalent to 2 mm. copper, 200 r daily for five days (total of 1,000 r in this first series). From the thirteenth to the seventeenth day, an additional 400 r was given with the same factors, at the rate of 100 r daily. On the twenty-ninth day from the beginning of treatment, the pain was almost completely relieved and she was able to get in and out of bed without assistance. On the sixtieth day, although the pain had been much improved the past few weeks, she had slight difficulty in getting out of bed in the morning. At the end of three and one-half months her weight had remained constant, she had lost one inch in height, and was able to change position without pain. She continued to have dull aching in the lumbar region and was unable to lift an average sized chair, showing that weakness was a factor in this instance. At this time additional treatment was given with the above factors at the rate of 100 r daily for six

days. At the end of four and one-half months the patient had lost three pounds in weight, no change in height, felt very much better, was able to go up and down steps without difficulty and stated, "pain is a thing of the past," and she was able to lift a chair above her head without difficulty. At the end of six months she had regained to her normal weight but had lost another one-quarter inch in height and complained of occasional backache for which a surgical corset gave relief.

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DISCUSSION

JAMES J. CLARK, M.D. (Atlanta, Ga.): I had the pleasure of hearing Dr. Merritt and Dr. Lattman's paper at the Detroit meeting, in 1935, at which time they discussed the x-ray treatment in hyperparathyroidism, and to-day's presentation is a valuable addition to the literature on the subject. I have found, myself, much trouble in convincing the average medical man that such a thing as hyperparathyroidism is responsible for the pain and the changes in the vertebræ which these patients show. For that reason, I have always felt that such papers as these should be read more often before a general medical assembly, if only to bring the condition to the attention of general practitioners so that they will have it in mind when we show them films of spines such as Dr. Caulk has demonstrated.

For a good many years, most of us have known or thought that there must be a close connection between these various types of bone lesions, such as osteitis fibrosis cystica or some of the osteomalacias, and the parathyroids. Also, certain forms of arthritis seem to be tied up quite closely to a too active function of the parathyroids.

However, I am certain that few men have paid the attention to parathyroid therapy by means of radiation that these stubborn diseases deserve. It seems highly probable that reabsorption of calcium from the bones in elderly persons may be due both to a low calcium intake and to hyperfunction of the parathyroids which take the calcium from the bony structure for use elsewhere in the body.

The authors' results in the treatment of such cases are striking, and certainly emphasize the importance of parathyroid therapy.

I had a patient, nine years old, who presented a history of several previous pathologic fractures of the femur, and was brought to me with a new fracture.

The x-ray examination was rather typical of osteitis fibrosis cystica, and she had multiple bone lesions elsewhere in the body. She had all the various laboratory and clinical tests and I may say that the laboratory tests were not entirely positive for parathyroid disease. However, her parathyroids were explored and they were found, according to the surgeon, to be normal. He would not remove them, although I tried to have him remove a few of them.

Radiation therapy of the parathyroid glands was begun as soon as the child was able to come to the x-ray department, and she has now gone five years without a fracture, and is growing and developing. She seems to be perfectly normal except for the deformities produced by her previous disease.

The left femur had had several fractures. X-ray examination showed various changes in the shaft; also, quite a change in the pubis ramus, broadening and thinning out, and a few early cysts could be seen in the opposite femur. The disease continued down the left leg, down the left tibia. The right tibia showed very little.

In the left foot tremendous changes had occurred in the metatarsus and the phalanges. There were also some changes in the bones of the opposite foot.

Since having parathyroid therapy and certain changes in her diet, she has been

practically free from symptoms for over five years.

Another of my patients was an elderly woman in whom, as Dr. Caulk said, it was nearly impossible to make a decent film of the spine. She had the fish-tail type of vertebra, narrowing down of the intervertebral space. She had a great deal of pain, with pronounced curvature above and lordosis below this vertebra.

The vertebra presented a ground-glass appearance, due to decalcification and demineralization. In these areas, it was possible to see the destruction or absorption of the body and the increase in width of the intervertebral spaces.

MILTON FRIEDMAN, M.D. (New York City): I have been greatly interested in the work of Merritt and Lattman, and now Merritt and Caulk, on the irradiation of the parathyroid glands for hyperparathyroidism from this point of view: A functioning adenoma of an endocrine gland or a hyperfunctioning endocrine gland is more radiosensitive than a non-functioning adenoma or a normal gland. We see the analogy in the pituitary gland, the normal pituitary gland having been known to withstand a total depth dose of 20,000 r extended over a period of several years. This occurred in a case which Dr. Kaplan is reporting shortly in a group of brain tumors. On the other hand, a hyperfunctioning pituitary gland at menopause can be inhibited with 1,200 r.

We see the analogy in the thyroid gland, in which the radioresistance of the normal thyroid gland, and the radiosensitivity of the hyperfunctioning gland are well known.

Therefore, it would be of interest to determine whether or not the parathyroid gland behaves similarly. A normal parathyroid gland is radioresistant; it would be of interest to demonstrate that a hyperfunctioning parathyroid adenoma is radiosensitive.

Hyperparathyroidism is accompanied by one rather consistent pathologic finding—high phosphatase in the blood. A disturbed calcium-phosphorus ratio is insufficient evidence upon which to base a diagnosis of hyperparathyroidism. It is usually necessary to demonstrate the presence of an excessive amount of phosphatase in the blood.

At the Hospital for Joint Diseases, we see a certain number of such cases and I continuously ask for the opportunity of irradiating the parathyroid glands. This has not been granted, and rightly so. The surgeon says, "It is so much quicker, so much easier, and so much simpler to remove this adenoma of the parathyroid gland than for you to irradiate and watch tentatively for the effect of radiation." One cannot refute that. That does not attempt to deny that we should investigate the question of whether or not a parathyroid gland is radiosensitive.

The case which Dr. Caulk demonstrated is an interesting one. Though the clinical and roentgen pictures suggest that this might be hyperparathyroidism, unfortunately, there is no phosphatase determination, and the diagnosis is not substantiated. Furthermore, after irradiation, there is no roentgen evidence of healing. Though this is not absolutely necessary, it is, nevertheless, important if one wishes to prove that the disease of hyperparathyroidism can be alleviated by irradiation of the parathyroid gland.

There is no doubt of the response of the symptoms to irradiation of the parathyroid gland, but I do not think we can call the disease hyperparathyroidism.

I wonder what the attendant bed rest and other general and local care might have contributed to it? Undoubtedly this factor has been taken into consideration; but, the only point I wish to make is that I do not think it has yet been demonstrated with any degree of authority that irradiation of the parathyroid gland has alleviated the lesions in the bone.

RICHARD C. NORTON, M.D. (Michigan City, Ind.): May I inquire what results, if any, may be expected from radiation treatment of the parathyroids in urinary tract calculi, which, as was mentioned, has

been proposed as one of this group of diseases due to hyperparathyroidism?

RALPH M. CAULK, M.D. (*closing*): I want to thank Dr. Clark and Dr. Friedman for their frank discussions. The questions which Dr. Friedman asked are continually arising. It is unfortunate that we have not been able to confirm this by analysis of the blood phosphatase, germ phosphatase, and blood calcium and phosphorus. However, authentic cases of osteitis fibrosa cystica have been reported by Ask-Upmark and Chauveau, with normal serum calcium.

This has also been found in experimental hyperthyroidism by Bodansky, Jaffe, and Shelling and the latter's collaborators. Similar relatively normal serum calcium concentrations in osteitis fibrosis were reported by Compere and Meyer-Borstel.

Be that as it may, we can at least approach the matter from an empirical basis. The condition I described does exist and it responds to x-ray therapy. There is no doubt about it. We can give the patient some relief. There must be many patients, such as I have described, all over the country, and, as radiologists, we certainly have an effective method of taking care of these cases.

As regards the calcification in the kidneys: we had no experience in treating parathyroids with this metastatic type of calcification.

SARCOID[1]

By ELLIS R. BADER, M.D., *Cincinnati, Ohio*

SARCOID, a disease which may involve the skin, bones, lymph nodes, and lungs, has been aptly described as "presenting clinical features of a chronic infectious granuloma persisting for years, sometimes spreading slowly from one organ to another, frequently relapsing, seldom producing serious constitutional symptoms, resistant to treatment, but at times healing spontaneously." The arsenicals and ultra-violet light are supposedly of value; however, some cases, such as the one which we are reporting, improve and clear up entirely without medication. Histologically, the lesion resembles tuberculosis, but there is no clinical tuberculosis, and all tests, including animal inoculations, prove negative. An atypical type of Hodgkin's disease has also been considered. The cases which have been reported as healed miliary tuberculosis are most probably sarcoid. The present consensus of opinion and the lack of definite causative findings place this disease in the category, "etiology unknown."

The case we are presenting is believed to be sarcoid with lung involvement. The patient, white male, aged 20 years, rather thin but not emaciated and not acutely ill, came to the hospital because of moderately enlarged glands in the neck, axillæ, and groins. He had had the glands for two years with no increase in size, no tenderness, no redness, or fluctuation. He complained of fatigue and loss of about twenty pounds in weight. There was no history of contact with tuberculosis, and he had never been engaged in a dusty occupation.

Physical examination of the lungs, as well as the remainder of the physical check-up except for the enlarged glands, was negative. Sputum, blood, urine, and feces were negative. The temperature range while the patient was in the hospital was 98 to 99°, pulse 80.

Histologic diagnosis of a gland excised from the groin was variously reported by numerous observers as endothelioma, tuberculosis, benign granuloma, and sarcoid.

[1] Read before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

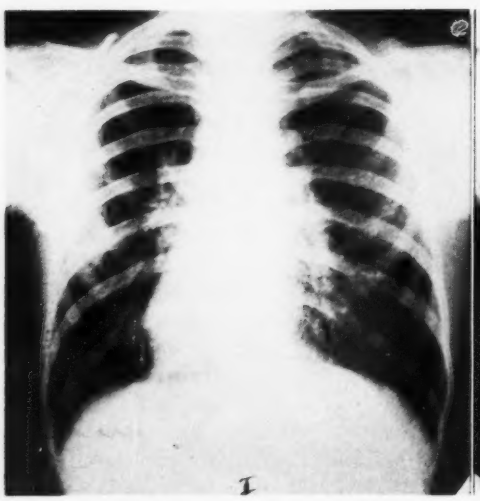


Fig. 1.

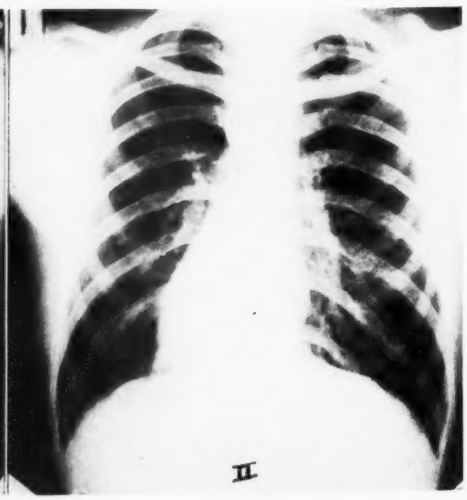
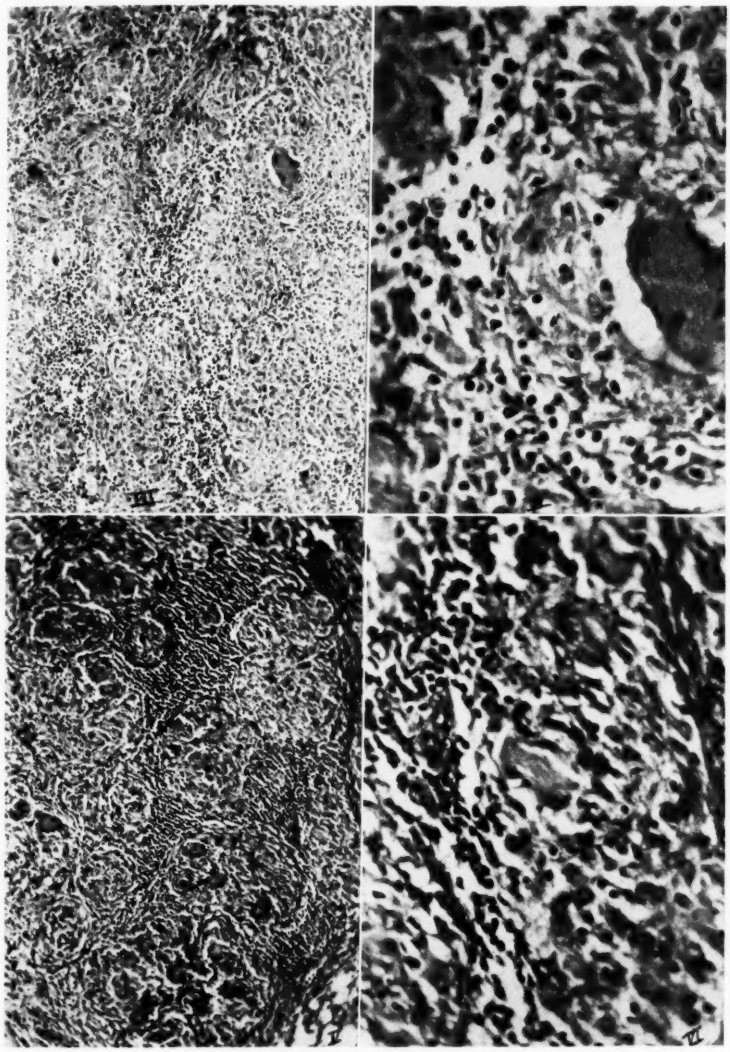


Fig. 2.

X-ray examination of the chest (Fig. 1), made July 2, 1936, showed floccular deposits in the parenchyma of the lung tissue, and increase in the hilus shadows and basal bronchial trunks. The findings resembled miliary tuberculosis. Pneumoconiosis could be ruled out since there was no history of a dusty occupation. Treatment was *nil* except for rest in bed at the hospital and later at home.

X-ray examination of the chest, Oct. 17, 1936, was completely negative for floccular deposits. There remained, however, a slight increase in the peribronchial tissue. The patient was completely well clinically.



Figs. 3 and 4 (*above*).
Figs. 5 and 6 (*below*).

X-ray examination (Fig. 2) of the chest on Feb. 11, 1937, showed no lung finding suggestive of pathology.

X-ray examination of the chest, made on

Jan. 14, 1939, showed no evidence of lung pathology.

Microscopic sections, high and low power (Fig. 3), showed proliferation of epithelioid cells and fibroblasts arranged in nests or whorls. Giant cells were present. There was no caseation as is seen in tuberculosis. Sarcoid is frequently diagnosed histologically as an atypical tuberculosis.

The final diagnosis was sarcoid, based on the microscopic section, x-ray chest findings, and clinical course of the disease (Fig. 4).

The second case had a similar history; however, the chest pathology has not completely cleared to date.

The patient, white male, 20 years of age, although seemingly fairly well nourished, gave a history of a loss of 34 pounds of weight. The chief complaint was loss of appetite and weight, also dry mouth. There was no particular discomfort. His blood pressure was 112/70; temperature 97.6°; pulse 70.

Physical examination was negative except for palpable anterior cervical and inguinal glands which were discrete, firm, not tender, and ranged in size from a small shot to a hazel nut.

The pathologist's report on the microscopic section (Figs. 5 and 6) of gland was as follows: "Section of lymph node. Complete replacement of the lymphoid tissue by a dense mass of compactly disposed rounded structures consisting of large central multinucleated giant cells and a periphery of large pale staining epithelioid cells. The individual structure is that of hard tubercles. These tubercles are so extremely numerous and so compactly placed that the general pattern closely simulates an epithelial malignancy, and it is surprising that in the presence of these countless compactly disposed tubercles there is virtually no caseation or necrosis. The pattern is that of proliferative or puerile tuberculosis. Never have seen this particular pattern of tuberculosis before." Subsequently, in conference with other pathologists, the pathologic diagnosis of sarcoid was made.

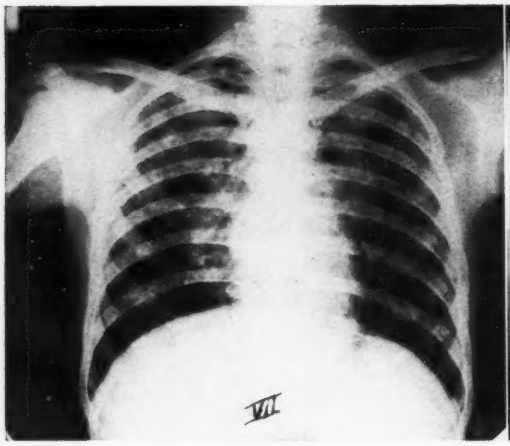


Fig. 7.

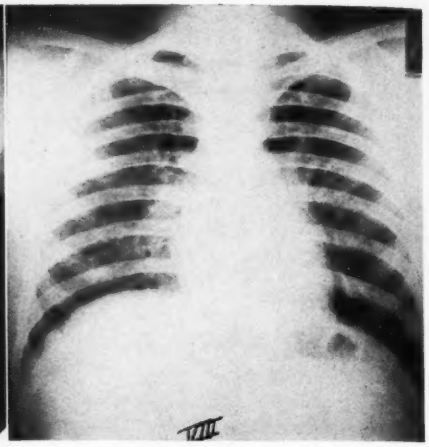


Fig. 8.

X-ray examination of the chest at the time of admittance to the hospital showed generalized increase in peribronchial tissue, with fairly discrete deposits in the lung parenchyma. I do not believe a definite diagnosis as to the exact nature of the lung pathology could at this time have been made, based solely upon the x-ray films (Fig. 7).

An examination of the chest made ap-

proximately two years later showed a definite decrease in the peribronchial density and lung infiltrate (Fig. 8).

The observation of the patient over this period showed a rapid return to normal, and although the chest is not entirely clear at the present time the patient would be considered clinically well. The diagnosis of sarcoid was in this instance again based on the biopsy findings, x-ray and clinical observation of the patient.

DISCUSSION

HOWARD P. DOUB, M.D. (Detroit, Mich.): Dr. Bader presented an excellent description of this unusual condition.

When Professor Boeck, of Norway, described this originally, he described it as a skin disease and it still shows the characteristic findings only in the skin. However, the changes are not always present in the skin. In one series, they were present in only two cases out of twelve. When the lesions are present in the skin, they are present as small nodules or large nodules or infiltration. There may be red, brown, or bluish areas of sharply demarcated skin lesions.

Diagnosis can be made only from a general clinical evaluation of all the factors present in the case. However, if the skin lesions are present, they will clinch the diagnosis in many cases.

Unfortunately, we do not know the etiology of this condition. It has been variously ascribed to tuberculosis, and other things—even to leprosy. I remember after I had described two of these cases in 1928, I had a letter from a man in South America who stated that these were not sarcoid; that they were cases of leprosy. However, we had carefully examined them, by smears, for leprosy, because we suspected that.

Darier-Roussy, however, studied the tuberculous etiology of this and while he could never demonstrate tubercle bacilli, after passing the examination through several series of animals, finally developed in these animals a tuberculous lesion.

Now that is not exact proof of the etiology, but it is somewhat suggestive. It has been said that it may be caused by a filterable virus.

The treatment is another thing about which we know nothing, because we do not know the etiology. So that from a general standpoint, we know the diagnosis of the disease but we do not know the etiology nor do we know the treatment of it.

METASTASIS TO BONE FROM CARCINOMA OF THE GASTRO-INTESTINAL TRACT[1,2]

By JUSTIN J. STEIN, M.D., *Hines, Illinois*

From the Surgical and Tumor Sections, Edward Hines, Jr., Hospital

A STUDY of bone metastasis from primary carcinoma situated in the gastro-intestinal tract reveals the incidence to be much higher than is generally thought. In Table I are shown the statistics based upon the findings in 1,005 cases of carcinoma of the gastro-intestinal tract which were admitted to Hines Hospital between Jan. 1, 1931, and Jan. 1, 1939. It is believed that the number would have been much higher if specific studies of bone metastasis in these cases had been made during hospitalization. Special roentgenologic study was made only in the cases in which there were complaints referable to the osseous system.

Esophagus.—In Müller's (1) series of 101 cases of carcinoma of the esophagus, metastasis to bone occurred in seven instances, or 6.93 per cent. Limacher (2) reported five from 119 cases of esophageal carcinoma; Clayton (3), two cases in a series of 41, and Willis (4), one in 12 cases.

Stomach.—Willis (4) stated that the incidence of skeletal metastasis from carcinoma of the stomach was approximately 5 per cent. In 1935, Kerr and Berger (5) reviewed the literature and found 143 cases of primary gastric carcinoma with metastasis to bone; they reported five additional cases. In a series of 309 cases of gastric carcinoma reported by Nisnjewitsch (6), bone metastasis occurred in eight instances (2.3 per cent). In Müller's (1) series of 309 cases of gastric carcinoma, bone metastasis occurred in eight cases, or 2.58 per cent. This figure corresponds very closely to the writer's series of 340 cases. Warwick (7) found metastasis to bone in four out of 176 cases of gastric carcinoma, or 2.2 per cent. Her findings were based on postmortem examination. Copeland (8) reported seven instances of bone metastasis in a series of 537 cases of gastric carcinoma. Sutherland, Decker, and Cilley (9) found 1.9 per cent bone metastasis from gastric carcinoma in a series of 1,032 cases of malignant bone metastasis. An incidence of 2.5 per cent is given by Matthews (10) and 5.7 per cent by Warren (11). Lawton (12) reviewed 606 cases of gastric carcinoma and found bone metastasis in only three. In a series of 903 cases reported by Winiwarter (13), bone metastasis was not present.

Because of the blood changes found in some cases of carcinoma of the stomach with bone metastasis, the illness may simulate pernicious anemia. Myeloblasts, myelocytes, erythroblasts, marked anemia, and a high color index occur in some patients following bone metastasis. There are many references to this blood picture in the literature.

Large and Small Intestine.—There are not enough cases of primary carcinoma of the small bowel in the literature to determine the real incidence of bone metastasis. In my series of 12 cases, bony involvement was present in only one. One case with bone metastasis has been reported by Ghormley and Valls (14); also one by

TABLE I.—INCIDENCE OF METASTASIS TO BONE FROM CARCINOMA OF THE GASTRO-INTESTINAL TRACT AT HINES HOSPITAL

Location of Primary Carcinoma	No. Cases	Cases with Metastasis to Bone	
		No.	Percentage
Esophagus	102	3	2.94
Stomach	340	9	2.65
Small intestine	12	1	8.33*
Colon, exclusive of rectum	216	2	0.93
Rectum	270	16	5.93
Pancreas	56	2	3.57
Gall bladder	6	1	16.66*
Ampulla of Vater	2	0	0
Bile duct	1	0	0

* Series of cases too small to determine true incidence.

[1] Accepted for publication in August, 1939.

[2] Published with the permission of the Medical Director of the Veterans Administration, who assumes no responsibility for the opinions expressed or the conclusions drawn by the author.

Fort (15). Ghormley and Valls also reported 10 cases with bone metastasis from a primary carcinoma of the colon (exclusive of rectum), or an incidence of 0.5 per cent in their series. Bone metastasis from a primary carcinoma in the colon occurred twice in the present series of 216 cases, or 0.93 per cent.

Rectum.—Nisnjewitsch (6) reported six cases of bone metastasis (10.5 per cent) in 57 cases of primary rectal carcinoma examined at postmortem. In a series of 117 rectal carcinoma cases reviewed by Aufses (16), there were eight instances (6.8 per cent) of bony involvement. Sutherland (17) reviewed 1,046 cases of bone metastasis and found 43 in which the primary lesion was in the colon. Of this group, 15 were located in the colon, six in the rectosigmoid, and 22 in the rectum. The diagnosis of bone metastasis was made by roentgenologic examination. Robertson (18) stated that he had found only four instances of carcinoma of the rectum or rectosigmoid in which metastasis to bone was present at postmortem examination, at the Mayo Clinic. He stated that, although a thorough survey of bones was not always possible at postmortem, his impression was that this complication was rare. Rosser (19) stated that he had never seen a proven case of metastatic carcinoma of the bone from a primary lesion in the rectum, in his cases. In Müller's (1) series, there were 57 rectal carcinomas from which metastasis to bone occurred in six instances, or 10.52 per cent. In my own series of 270 cases, bone metastasis occurred in 16 cases, or 5.93 per cent. Histologic or postmortem proof was obtained in 12 of the 16 cases. It is interesting to note that, after several cases of bone metastasis from carcinoma of the rectum were seen, routine pelvic roentgenograms were made in a small series of cases, and, surprisingly enough, bone metastasis to the pelvis was found in which no clinical symptoms were present. It would be of value to the surgeon, especially from a prognostic standpoint, if roentgenograms of the pelvis, chest, and lumbar spine were made in cases of rectal carcinoma in which radical surgical procedures are contemplated.

Accessory Gastro-intestinal Organs.—There were 56 cases of pancreatic carcinoma in the present series in which bone metastasis occurred twice, or 3.57 per cent. Limacher (2) found one case in 21 postmortems in which bone metastasis was present. In Müller's (1) series of 19 cases of primary pancreatic carcinoma, the incidence of bone metastasis was 21.05 per cent. He also noted bone metastasis in three cases (20 per cent) of 15 primary liver carcinomas. In Schlesinger's (20) study of 54 cases with metastatic growths in the vertebræ, the primary tumors were located as follows: breast, 10; esophagus, 9; thyroid, 9; uterus, 6; bronchus, 5; stomach, 4; prostate, 3; gall bladder, 2; ovary, 1; sigmoid, 1; rectum, 1; kidney, 1, and pancreas, 1. In a review of the literature, in 1924, Jenkinson (21) found a total of 41 cases having bone metastasis. The primary lesions were located as follows: stomach, 32; gall bladder, 3; rectum, 2; bile duct, 1; appendix, 1; pancreas, 1, and colon, 1. Occasionally, individual case reports are seen in the literature of carcinomas of the pancreas, liver, bile ducts, etc. One case of primary carcinoma of the gall bladder in this series had bone metastasis.

Sites of Bone Metastasis.—Of the 153 instances in which the affected bone was mentioned in the series of gastric carcinoma with bone metastasis studied by Kerr and Berger (5), the bones were involved in the following order: spine, 28.8 per cent; ribs, 19 per cent; femur, 15.7 per cent; sternum, 9.8 per cent; pelvis, 7.2 per cent; humerus and skull, each, 5.85 per cent; sacrum, 2.6 per cent; clavicle, tibia, scapula, each, 1.3 per cent; radius, 0.65 per cent, and the entire skeleton, 0.65 per cent.

The bones most frequently involved, in a study of 24 cases of rectal carcinoma with bone metastasis made by Aufses (16), were as follows in the same order of frequency: vertebræ, femur, ribs, skull and

sternum, humerus, pelvis, sacrum, radius, scapula, ulna.

In Moore's (22) study of 65 cases of metastasis to bone from any organ, the bones were involved in the following order: spine, 22; pelvis, 11; femur, 9; ribs, 6; humerus, 6; radius, 3; skull, 3; tibia, 2; sternum, clavicle, and bones of hand, 1 each.

In a review of 1,625 cases of metastatic carcinoma to bone from the following organs, breast, prostate, lung, thyroid, stomach, kidney, colon, and uterus, the five most commonly involved bones were the spine, pelvis, ribs, femur, and skull.

In a study of the cases in this series, the size, degree of malignancy, and the location of the primary tumor with reference to the involved organ had no bearing on the probability of metastasis to bone. If bone metastasis takes place, the primary lesion may be of either low or high degree of malignancy; it may be small or large in size.

Metastasis to bones takes place by both the hematogenous and lymphogenous routes. Probably most of the cases occur by the hematogenous route, since carcinoma cells can be found in the bone marrow vessels. Von Recklinghausen was of the opinion that the rich capillary network of bone and also the sluggishness of the blood in the capillaries accounts for the bone involvement.

Roentgenographic Appearance.—The osteoclastic form of metastasis which is seen most frequently secondary to carcinoma of the breast is also characteristic of most of the cases of bone involvement from primary lesions in the gastro-intestinal tract. In some of the series reported, the osteoplastic form of metastasis was also present. However, in the present series the predominant picture was of the osteoclastic type. Occasionally a lesion would have both osteoclastic and osteoplastic elements and it would be difficult to determine the exact nature.

SUMMARY

1. The incidence of bone metastasis from primary carcinoma of the gastro-intestinal tract, although uncommon, is higher than is generally thought.

2. By far the majority of the metastatic bone lesions from the gastro-intestinal tract are of the osteoclastic type.

3. In a review of 1,625 cases of metastatic carcinomas to bone, the five most commonly involved bones were the spine, pelvis, ribs, femur, and skull.

4. The probability of bone metastasis cannot be determined by the degree of malignancy, size, age, or location of the primary tumor.

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GRAPHIC CONSTRUCTIONS FOR LOCALIZATION, MENSURATION, AND REDUCTION: STANDARD SYMBOLS AND NOMENCLATURE[1]

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FOR simplicity, we have divided this treatise into four sections, as follows:

I. Introduction.
II. Standard symbols, standard nomenclature, and basic constructions.
III. Practical application.
IV. Conclusion.

I.—INTRODUCTION

We have written this article for two reasons: first, to record basic graphic constructions to be utilized in roentgenographic localization and mensuration, and, second, to introduce standard symbols and nomenclature in order to simplify the reading and writing of papers on these and related subjects.

We wish at this point to state that all the constructions advocated are original except the "vector image intersection" for projection determination (Fig. 5-*A*) and the "space construction" for mensuration (Fig. 6).

The determination of projection by the offset or vector image reduction methods is extremely valuable, for by these methods we are able to avoid the eccentric exposures (1, 2), which are otherwise unavoidable. Eccentric exposures are adequate for measuring purposes, but are valueless for diagnostic purposes, since they represent oblique views. Any constructions which obviate the necessity for eccentric focal positioning are of inestimable value. By the methods which we suggest it is possible for the focal positions to be directly over, or in line with, the points to be measured or localized.

II.—STANDARD SYMBOLS, STANDARD NOMENCLATURE, AND BASIC CONSTRUCTIONS

The standard symbols and nomenclature are described in Figures 1-*A* and 1-*B*. The basic constructions are described in Figures 2 through 6, as follows:

(*A*) Depth determination by standard depth curve (Figure 2-*A*), and by depth localization rectangle (Figure 2-*B*).

(*B*) Line reduction by reduction triangle (Figure 3).

(*C*) Plane reduction by radial reduction (Figure 4-*A*) and by perimetric reduction (Figure 4-*B*).

(*D*) Projection determination by vector image intersection (Figure 5-*A*), by the offset method (Figures 5-*B* and 5-*C*), and by the vector image reduction method (Figure 5-*D*).

(*E*) Mensuration by space construction (Figure 6).

III.—PRACTICAL APPLICATION

The acceptance of the symbols, nomenclature, and constructions advocated in the above section will immeasurably simplify the reading and writing of treatises dealing with localization and mensuration. We will demonstrate how this is accomplished by the following hypothetic case, in which the entire procedure is described simply and directly, without so much as a single drawing or illustration.

The patient, Mrs. M., is at term, and we desire to measure the pelvic diameter extending from the tip of the left ischial spine (hereinafter designated 3) and the tip of the coccyx (designated 4). The patient lies on her back in the center of

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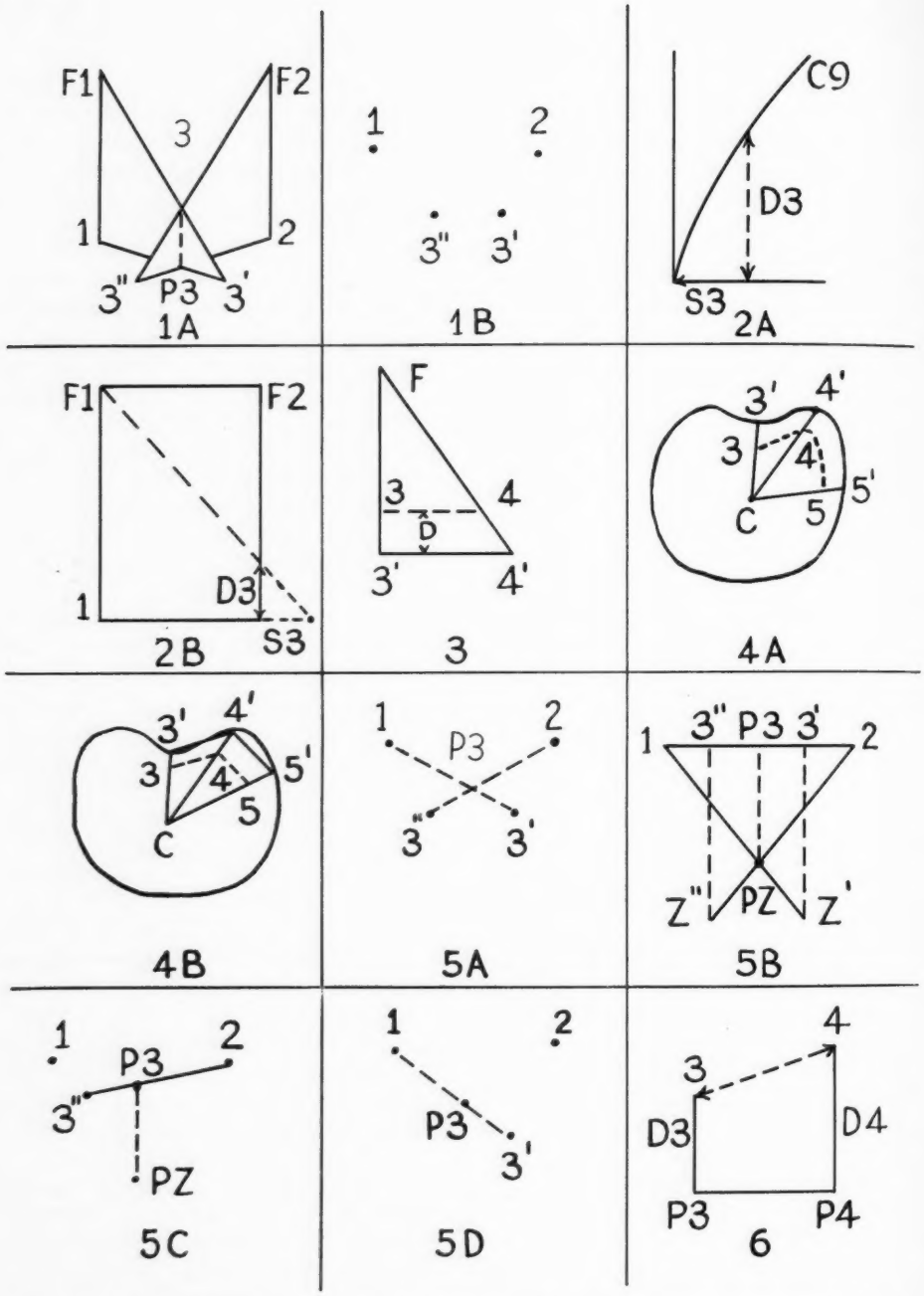

Fig. 1-*A*. *F* 1, *F* 2 are the focal positions 1 and 2; 1 and 2, the perpendicular projections of *F* 1, *F* 2 on roentgenogram, *X*. If more than one roentgenogram is made, *X* 1 refers to *F* 1 exposure, whereas *X* 2 refers to *F* 2 exposure. The figure 3 represents any point in space. If more than one point is involved they are called successively 4, 5, 6, etc. Note that 1 and 2 are reserved for focal positions or their projections. The numerals 3′ and 3″ are images of 3 from *F* 1 and *F* 2; *F S* is the distance *F* 1–*F* 2 (usually 3, 6, or 9 inches); *S* 3 equals 3′–3″ (image shift); *H* is the perpendicular distance from *F* 1, or *F* 2 to *X* (usually 32 inches); *P* 3 is the perpendicular projection of 3 on *X*; *D* 3 equals perpendicular distance from 3 to *X*.

the x-ray table. *F* 1 is centered over the umbilicus; *H* equals 32 inches; *X* 1 is exposed. *F* 2 is 9 inches below *F* 1; *X* 2 is exposed. Mark 3′, 3″, 4′, and 4″ on *X* 1 and *X* 2. Transcribe *X* 1 and *X* 2. Proceed as follows: Measure *D* 3 and *D* 4 by applying *S* 3 and *S* 4 from transcription in standard depth curve, *FS* equals 9 inches, *H* equals 32 inches; locate *P* 3 in transcription by vector image intersection; locate *P* 4 in transcription by the offset or vector image reduction methods; measure *P* 3—*P* 4 in transcription, and construct line 3–4 in space by space construction method, using *P* 3–*P* 4, *D* 3, *D* 4 as knowns.

IV.—CONCLUSION

In localization and mensuration, we have strongly advocated standard factors of exposure (3, 4, 5). The factors advocated by us have been *H* equals 32 inches, *FS* equals 3, 6, or 9 inches.

Drummond and Schmela (6) recognize the desirability of using standard factors of exposure, and they employ factors identical with ours. We now advocate the adoption of standard symbols and nomenclature to facilitate the reading and writing of articles dealing with localization, mensuration, and related subjects.

It is obvious that the reduction methods advocated by us are applicable to structures lying in planes parallel to the plate (4), namely, natural anatomic planes, artificially created planes (4), planeographically determined planes (3, 4), and planigraphs.

We strongly recommend localization and mensuration by the graphic method. We believe it is superior to other methods because it is simple, speedy, and accurate; it requires no instrumentation, no computation, and no reference tables; it affords a permanent and available record for inspection and correction, if need be; it shows the relationship of points more readily than records based on linear meas-

Fig. 1-*B*. This is a transcription (copy) of the points demonstrated on *X* (Fig. 1-*A*), except that only the points necessary for localization or mensuration are copied.

Fig. 2-*A*. Depth by standard depth curve. *C* 9 is the standard depth curve for *F S* equals 9 inches, *H* equals 32 inches; *S* 3 is applied along the horizontal ordinate and *D* 3 is measured along the appropriate vertical ordinate.

Fig. 2-*B*. Depth by localization rectangle. The depth localization rectangle is constructed with *F S* as one dimension and *H* as the other. *S* 3 is applied along the *F S* dimension and an oblique line intersects the *H* dimension so that *D* 3 is measurable.

Fig. 3. Linear reduction by reduction triangle. Given a line 3′–4′, the image of line 3–4 which lies parallel to the film, and distance *D*, above it, the object is to construct 3–4 from image 3′–4′. The method is as follows: 3′–4′ is larger than 3–4. By reducing 3′–4′ to its proper size, 3–4 may be obtained. For this purpose we make use of the reduction triangle, which is a right-angled triangle the vertical arm of which is *H*, and the horizontal arm 3′–4′. A second line drawn parallel to 3′–4′, and depth, *D*, above it, intersects the vertical arm in 3, and the hypotenuse in 4, line 3–4 representing the line in question.

Fig. 4-*A*. Radial reduction of planes. 3′–4′–5′ is the image of a plane 3–4–5, in space parallel to the film, and distance, *D*, above it. To reconstruct plane 3–4–5: from any point *C* draw radial lines *C*-3′, *C*-4′, *C*-5′, etc. Reduce each line in the reduction triangle, thus locating points 3, 4, 5, etc., which connect by appropriate lines.

Fig. 4-*B*. Perimetric reduction of planes. Connect points 3′–4′, also 4′–5′, etc. Locate 3 by radial reduction, draw a line 3–4 parallel to 3′–4′, locating point 4 in this manner. Continue this process, locating 5, etc.

Fig. 5-*A*. Vector image intersection for projection determination. In transcription (see Fig. 1-*B*) draw vector images 1–3′ and 2–3″. They intersect at *P* 3, the projection of 3.

Fig. 5-*B*. Offset method for projection. When 3′ and 3″ fall along 1–2, the vector images 1–3′ and 2–3″ do not intersect but overlie, so that *P* 3 cannot be determined by this method. To determine *P* 3 under such circumstances, draw lines 3′-*Z*′ and 3″-*Z*″ perpendicular to 1–2, and equal to each other. Locate *P Z* by intersection of lines 1-*Z*′ and 2-*Z*″. Project *P Z* on 1–2 at *P* 3.

Fig. 5-*C*. Offset method for projection. When 3′ and 3″ lie close to 1–2, proceed as in Figure 5-*B*, but project *P Z* on either vector image, 1–3′, or 2–3″ to locate *P* 3; *P Z*–*P* 3 is perpendicular to 1–2.

Fig. 5-*D*. Projection determination by vector image reduction. Points 3′ and 3″ are images of 3 which lies *D*3 above *X*. To determine *P* 3, measure vector image 1–3′ (or 2–3″); reduce 1–3′ in reduction triangle. The reduced line is equal to 1-*P* 3, which lay off along vector image 1–3′, thus locating *P* 3.

Fig. 6. Space construction for mensuration. Given line 3–4 in space, so that 3 is *D* 3 above plate and 4 is *D* 4 above plate, to measure 3–4, determine *P* 3 and *P* 4, and measure this distance. At *P* 3 erect a perpendicular equal to *D* 3; at *P* 4 erect a perpendicular equal to *D* 4. The free ends of the perpendiculars represent points 3 and 4 as they actually exist in space. The line joining 3–4 is the measure of the line in question.

urements, and it may be of great value to the surgeon in directing surgical procedures (2).

Finally, may we add that although we do not claim originality for the determination of length by the "space construction method," we actually devised such a construction as early as 1919 (7).

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CASE REPORTS AND NEW DEVICES

SPONTANEOUS GASTRO-ENTEROSTOMY[1]

REPORT OF CASE

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Gastrojejunal fistula, of spontaneous origin, must be extremely rare, as a search through the literature, since 1923, fails to reveal such a case. Fistulæ between any of the segments of the alimentary tract are relatively uncommon. Gastrojejuno-colic fistula is by far the most frequent; Fardelmann (1) found 229 cases up to 1930, and there have been several reports of such cases each year since then. These fistulæ usually follow gastro-enterostomy performed in the treatment of peptic ulcer and its complications, but occasionally result from spontaneous perforation by carcinoma. Frank (2) collected 16 cases of gastroduodenal fistula, recognized by x-ray, due to perforation of ulcer or carcinoma, adding one of his own. Spontaneous ileo-ileostomy, ileocolostomy, and gastrocolostomy have also been reported. The case here presented was found to be a perforation of a gastric carcinoma into the proximal jejunum. Because of its rarity, and because there was opportunity to study this lesion roentgenologically, gastroscopically, and at operation, we feel justified in making this brief report.

Report of Case.—J. B., 58-year-old coal miner, was first seen in the clinic on April 7, 1939, complaining of epigastric and left upper quadrant pain, a lump in the left upper quadrant, nausea, and low back pain. About nine months before, rather suddenly, he began to awaken in the morning with pain in the epigastrium, radiating to the left upper quadrant. This persisted throughout the day, without any observed relation to food, drink, or any bodily activity. Shortly thereafter he became conscious of a small lump in the left upper quadrant which was hard and moderately tender. He believed that this lump had not increased significantly in size since it was first noticed, but that it had become harder. His appetite became poor and he began to lose weight, this loss reaching 20 pounds at the time of his first visit to the clinic. Six months before admission he suffered from increasingly distressing nausea, but at no time did he vomit. He also had low back pain, having no relation to activity, which had become much more severe recently. He had never observed tarry stools, and his bowel status remained normal throughout his illness.

Fig. 1. Roentgenogram of barium-filled stomach. Arrow shows point of perforation.

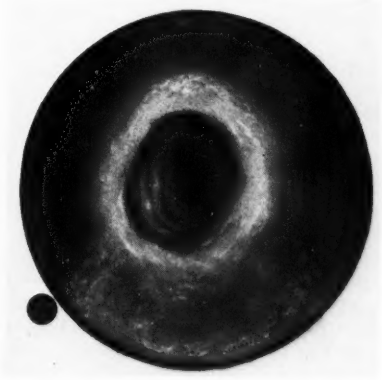

Fig. 2. Gastroscopic appearance of carcinoma, as one looks into a loop of the jejunum.

On physical examination the patient was obviously pale and undernourished. The skin was dry and loose. There were moderate ar-

[1] Accepted for publication in November, 1939.

teriosclerotic changes seen in the vessels of the ocular fundi and the peripheral vessels were somewhat thickened. The tongue was patchily coated, not red or smooth. Heart and lungs were negative to examination. Just above and to the left of the umbilicus there was easily palpable a hard, rounded, irregular, tender mass approximately 8 cm. in diameter, which was almost completely fixed to the posterior parietes.

Laboratory investigation showed a hypochromic anemia, with 3,250,000 erythrocytes per cu. mm. and hemoglobin 54 per cent (Sahli). The total leukocyte count was 15,700 cells per cu. mm., and the hemogram 4 per cent juveniles, 9 per cent stab cells, 70 per cent segmented cells, 12 per cent lymphocytes, and 5 per cent large monocytes. The blood Kahn test was negative. N.P.N. 20 mg. per cent. Guaiac test on the stool was 4+.

Gastro-intestinal x-ray examination showed (Fig. 1): Divulsion of the esophageal cardia; complete occlusion of the prepyloric stomach, this defect being directly posterior to the abdominal mass by palpation; passage of the barium meal from the greater curvature of the stomach directly and without delay into the jejunum; adequate gastric emptying. The picture was almost exactly that seen after partial gastric resection of the stomach and gastroenterostomy.

Roentgen kymograms of the stomach, duodenum, and jejunum, with the stomach partially filled with barium, were interpreted by Wendell G. Scott, M.D., of the Mallinckrodt Institute of Radiology, as follows: "Peristaltic activity is noted over the proximal portion of the greater curvature in which one large peristaltic wave is recorded. There is no evidence of peristaltic activity in the dependent portion of the fundus, nor in the pyloric portion of the stomach containing the barium. Very small serrated movements on the border of the stomach and the small intestine are caused by the transmitted movements of the arterial system. Hyperactive peristalsis is seen in both loops of the small intestine. The absence of peristalsis of the lesser curvature of the stomach suggests that this area is infiltrated by the lesion. Similarly, the presence of active peristalsis in both loops of intestine show that these structures are involved over only a small area."

On gastroscopic examination (Fig. 2) there was visualized on the posterior greater curvature area of the pars media a lesion roughly elliptical in contour, of a dirty grayish color. A receding dark brown spot, apparently the fistulous stoma, was seen in one portion of this. The mucosa of the anterior wall was pale and ischemic. The surrounding mucosa, especially on the posterior wall, appeared to have an irregular swollen topography which was mammillated, and was interpreted as an old atrophic gastritis.

At operation, on April 22, 1939, Franklin Walton, M.D., reported the following: "There were perhaps 300 c.c. slightly blood-tinged free fluid. There was a malignant ulcer involving the posterior aspect of the stomach at the junction of the middle and lower thirds. This had perforated into the lesser sac and involved the pancreas, and it was attached to a loop of proximal jejunum. There was, in addition, a kissing ulcer that grossly was malignant opposite this original lesion in the anterior aspect of the stomach. No further attempt was made to ascertain the mechanism by which the loop of intestine had entered the lesser sac but it was evident that this had occurred at the right of the middle colic artery, through either a congenital opening in the posterior leaf of the transverse mesocolon or in some similar fashion. There were several nodules in the left lobe of the liver which were obviously malignant, but the regional glands were free from involvement grossly. Nodule in liver removed for microscopic section."

Microscopic examination of this nodule revealed a typical adenocarcinoma of the gut.

DISCUSSION

A search of the literature since 1923 fails to reveal the report of a similar case of spontaneous gastro-enterostomy. The mechanism by which this fistula occurred is hypothetical. Two possibilities present themselves; either erosion posteriorly through the lesser peritoneal sac into the greater cavity with fixation and subsequent perforation of the proximal jejunum, or herniation of the small bowel through a congenital foramen with fixation by the neoplasm.

It is noteworthy that such a lesion could be shown both roentgenographically and by gastroscopy. The former method revealed the anatomic relationships and the pathologic physiology, and by these predicated the nature of the lesion. The gastroscope revealed the neoplasm directly and also showed the fistulous stoma. One could not have determined the exact intestinal segment involved by this latter diagnostic method alone. Kymography further elucidated, by altered motility of the involved portions of the stomach and intestine, the extent of the pathologic infiltration.

We feel fortunate in having had the opportunity to study by several modern methods this gastro-enterologic rarity, in which all that could have been desired from surgical palliation was providentially accomplished.

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CHONDRODYSTROPHY[1]

PRENATAL DIAGNOSIS POSSIBLE

By E. J. THALHEIMER, M.D., *and* J. GERSHON-COHEN, M.D., *Philadelphia*

With the increasing use of pelvicephalometric roentgenography, it is to be expected that there will be many fetal anomalies recognized. This observation has already been made by many, notably Doub (2), Cronberg (1), Moloy and Swenson (3). Anencephaly, hydrocephalus, and spina bifida have been diagnosed many times before birth, but, so far as we can determine, Cronberg is the only one to have reported a case of achondroplasia diagnosed by x-ray examination before birth. Our case was first diagnosed six months after delivery, but review of the films made for pelvicephalometry reveals all the changes in the bones from which a prenatal diagnosis might have been made.

Case Report.—When this child was six months old, a lumbar kyphosis was first noticed by the mother. X-ray examinations made at this time revealed the presence of achondroplasia (chondrodystrophy).

No familial occurrence of achondroplasia was found.

The physical examination of the patient revealed that the extremities were shorter than normal for the length of the body. The head seemed enlarged, with slight depression of the nasal bridge.

At seven months of age, the child's weight was seventeen pounds, eight ounces; height, twenty-four and one-half inches; sitting height, sixteen and one-half inches; circumference of head, nineteen inches; chest, sixteen inches; abdomen, sixteen inches; length from the acromion to the external humeral condyle, the arm measured four and one-quarter inches; from the lateral condyle to the ulnar styloid, three and three-quarter inches; from the antero-superior spine of the left ilium to the external malleolus of the fibula, nine inches. The Wassermann test was negative; the blood smear and counts at birth were not abnormal.

Fig. 1. The lumbar kyphosis is evident in the lateral view of the spine. The margins of the lumbar vertebral bodies are irregular; the costochondral junctions are deformed and widened, the shafts of the long bones are short, wide, and flared at the ends.

[1] Accepted for publication in October, 1939.

The x-ray examinations disclosed shortening of all the long bones, with widening of the medulla and thinning of the cortex, irregular widening and flaring of the ends of the shafts, increased spacing between the vertebral bodies, especially in the area of lumbar kyphosis,

Fig. 2. In this view, the deforming changes are demonstrated at the ends of the long bones, especially in the knee and elbow.

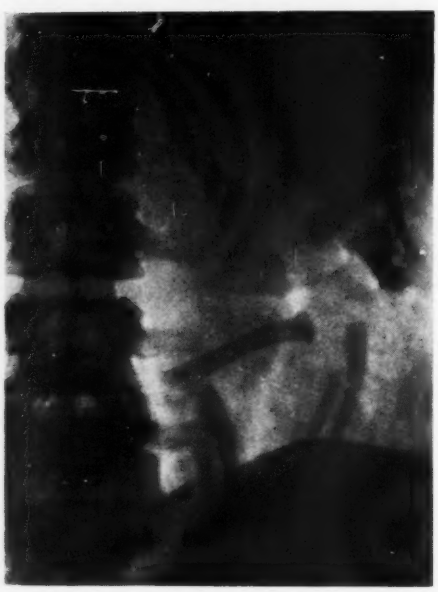

Fig. 3. The short, wide bones with deformed ends, changes characteristic of chondrodystrophy, are visible in the fetus *in utero*.

flaring of the rib ends, and flattening of the acetabula (Figs. 1 and 2). In the prenatal films one may discern the irregular flaring of the ends of the bones, the shortened shafts of the long bones with their wide medullary spaces and thin cortices (Fig. 3).

Summary.—A case of chondrodystrophy is reported, diagnosed when the patient was six months old. The diagnosis might have been made before birth, with the fetus *in utero*, as demonstrated in this case report. Chondrodystrophy is thus another abnormality that may be diagnosed roentgenographically before birth.

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CASES OF HYDROPNEUMOTHORAX, REMOVED INTACT WITH ENTIRE PLEURAL SAC[1]

By CHARLES GOTTLIEB, M.D., Radiologist, *and* MILTON MALEV, M.D., Pathologist, *New York City*

From the Riverside Hospital

In cases in which the parietal pleura is thickened from longstanding pneumothorax, with or without fluid, it is sometimes possible to remove the entire pleural sac intact, preserving the hydropneumothorax. This was done in the two cases here summarized. If the original costal incision does not penetrate the inflated pleura, it can be stripped away from the chest wall with comparative ease.

[1] Accepted for publication in October, 1939.

Case 1. A. M., 58-year-old negro woman, houseworker, admitted to Riverside Hospital on March 3, 1938, acutely ill, emaciated, and markedly toxic. She had a known history of pulmonary tuberculosis for the past year during which time she had been to three city hospitals. Ten months before admission she had had a bilateral pneumothorax, and for six months had come into a clinic for refills. She was then hospitalized because of weakness, and a left pneumothorax was discontinued as in-

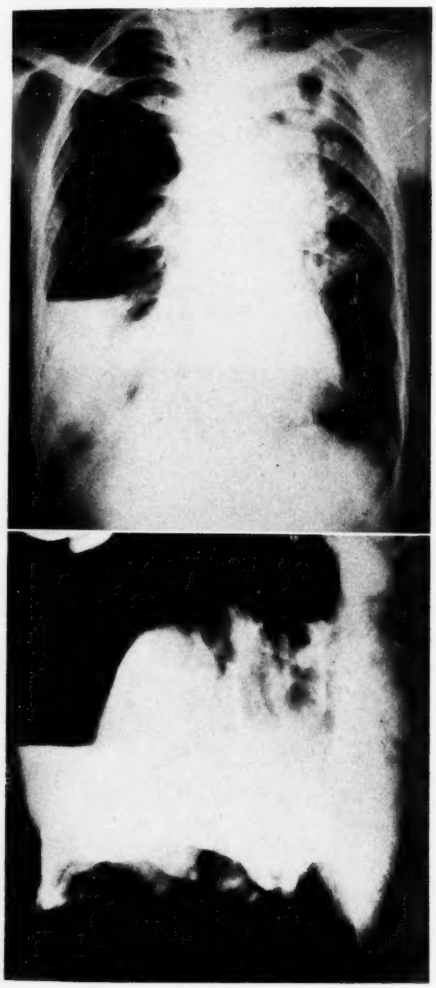


Fig. 1-*A* (*upper*).
Fig. 1-*B* (*lower*).

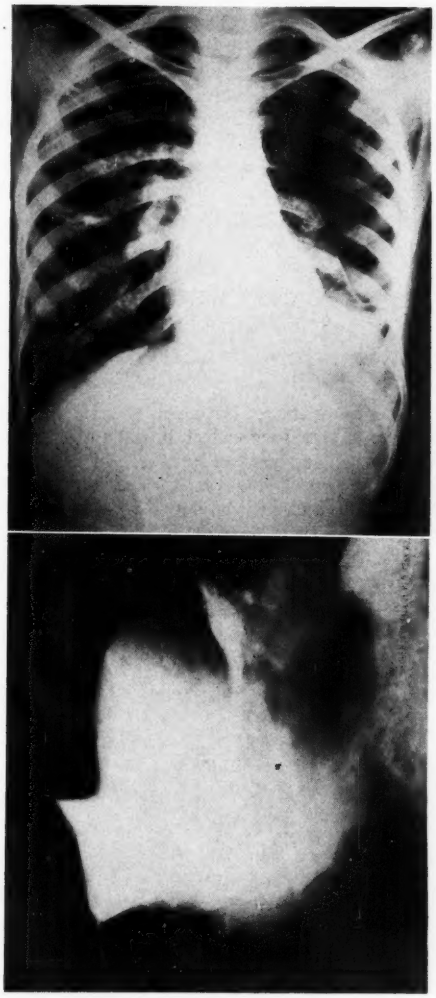


Fig. 2-*A* (*upper*).
Fig. 2-*B* (*lower*).

effective. Three months before admission here she developed fluid in the right chest, which was repeatedly removed and replaced with air. Her two weeks' course in this hospital was progressively down hill and she received no active therapy.

Anatomical Diagnosis.—Pulmonary laryngeal and intestinal tuberculosis; right hydropneumothorax; generalized amyloidosis; uterine fibroids.

Case 2. M. B., 24-year-old negro woman, admitted to Riverside Hospital on July 28, 1937. She had a three months' history of typical symptoms and had spent some time at another hospital where a left pneumothorax was done and several refills given. During her stay at this hospital (two months) she received small weekly refills with negative pressures, and fluid was removed occasionally as it accumulated.

Complications.—Severe tuberculous enterocolitis and peritonitis which caused her death.

Anatomical Diagnosis.—Pulmonary tuberculosis with left hydropneumothorax; intestinal tuberculosis; plastic tuberculous peritonitis; mucocele of the appendix.

RADIUM BOMB TO UTILIZE VARIOUS APPLICATORS[1]

By W. E. PENNINGTON, M.D., *Indianapolis, Indiana*

The presentation of a bomb as an applicator is not a new idea for it has been utilized on numerous occasions. The purpose of this device is to protect structures which do not need radiation and to assist even distribution of the dosage.

Almost everyone who has been using radium over a period of years has accumulated applicators of various sizes. There is considerable difficulty in applying these different packages evenly over a given surface.

The larger bomb in this presentation is made up of 3 mm. of brass for the filter. The side wall of the chamber is also ground to 3 mm. of brass; however, this wall may be thicker if more protection is needed. The floor is 5 mm. of brass. The spool-shaped stem is composed of aluminum and reduces the total weight which the floor of the vagina must support. The transverse diameter and the long axis of this model can be varied according to the size needed for any particular patient. The bomb

[1] Accepted in June, 1940.

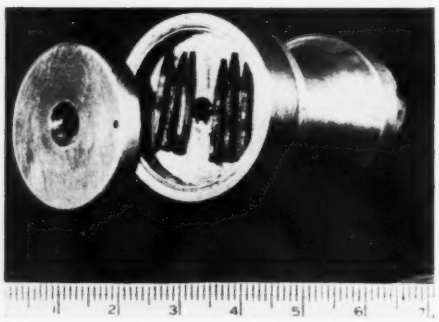


Fig. 1. Inside view of bomb compartment which accommodates needles and capsules.

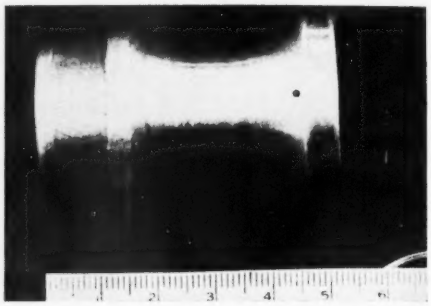


Fig. 2. Side view of Figure 1 showing copper head and aluminum spool.

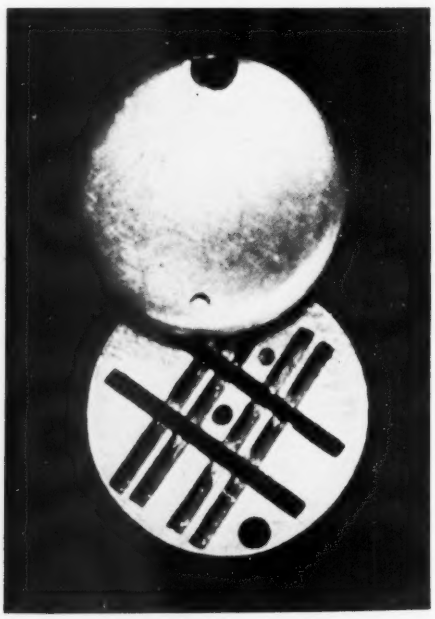


Fig. 3. Showing inside of bomb to accommodate needles and to show maximum copper protection for vaginal walls.

has a long axis; therefore, it will not rotate and it will stay in the position in which it is placed. Its spool shape will permit packing gauze about the small part of the shaft and consequently helps to stabilize the position desired.

It is not unusual to see undesirable changes in the vaginal canal which are remote from the involved tissue. Blanching of the vaginal wall has been observed up to the peritoneum. A common method of applying radium about the cervix by means of a plaque or by the application of multiple tubes frequently gives bladder and rectal symptoms, because these structures have not been sufficiently protected. Stenosis of the vagina does not occur as frequently with this type of applicator as it does with the use of gauze packing.

The writer has frequently used barium plugs against the radium which have markedly decreased inflammatory reactions in the vagina, bladder, and rectum; however, a barium plug is retained with difficulty because of its disposition to slip under pressure.

In conclusion, the following facts may be enumerated to commend the applicator:

1. Proper filtration of the rays may be effected.

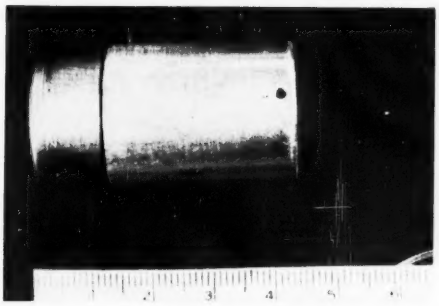


Fig. 4. Side view of Figure 3.

2. Normal structures such as the bladder, vaginal walls, and rectum are protected.

3. A constant position of the applicator may be maintained.

4. Intra-uterine radiations are not precluded.

5. These applicators direct treatment to the cervix in essentially one plane and coincidentally protect tissues which do not need radiation.

214 Hume Mansur Building

PRESIDENTIAL ADDRESS[1]

The American College of Radiology to-day is functioning as most colleges: in other words, it is representing organized radiology in all its public relations, economic and general problems that face the radiologist.

The presidential addresses for the past several years have dealt largely with the many unfair tactics used by numerous hospitals and medical groups in connection with radiologic problems. This has been entirely correct; much has been accomplished and much still has to be accomplished along this line.

Thomas Hughes said, "There are two kinds of discontent in this world; the discontent that works and the discontent that wrings its hands. The first gets what it wants and the second loses what it has." We shall ever be indebted to the men who have, in addition to their scientific attainments, been unwilling to wring their hands but have spared neither time, effort, nor money in bringing radiology to its present position in medicine.

A letter of welcome to the large number of new members who came into the Society this year brought many interesting replies. They were mainly of three types: The majority wish to co-operate and help the College in any way possible to accomplish its undertakings; others mentioned specific instances of bad ethics on the part of a hospital or radiologist and wished to know what, if anything, the College could do about it; still others cited instances in their own cities in which a radiologist of national reputation was doing things which were not in the best interest of radiology, the medical profession, or the hospital and though the College was aware of these facts, it was unable to do anything about it.

My remarks will be limited largely to a discussion of these letters and a discussion of the obligations of a radiologist to the public, the medical profession, the hospital, and radiology as a specialty. Later I would like to make a few suggestions as to the manner in which I believe we can help the College to accomplish more thoroughly its objects in view. I realize fully that a few radiologists in this country who have never encountered any trouble with their hospitals feel that good scientific work is all that is necessary for radiology to continue to succeed. But most of us, even though we have had no trouble, are convinced that we must be well organized and work together if radiology is to be spared the fate of pathology. Theodore Roosevelt said, "Every man owes a part of his time and money to the profession in which he is engaged."

The first group of letters from individuals who offered to co-operate in every possible way needs no further discussion than the thanks and appreciation of the College.

The communications in group two gave instances of bad ethics on the part of a hospital or radiologist and wished to know what, if anything, the College could do about it. The College has in many instances been able to help materially such situations. In other instances there have been many reasons why the College could accomplish little. In the first place, bad feelings among radiologists in a city prevent their co-operation in dealing with any hospital trouble.

The third group of letters cited instances in their own cities where prominent radiologists connected with large institutions gave little service to the hospital. This is again a difficult thing for the College to handle. Examples are cited concerning prominent men who head the departments of radiology in large institutions but who give very little of their time to these institutions. They will visit the department two or three times a week and read a few films. Interns do the fluoroscopy. Between their visits, the interns and members of the staff interpret the films. This type of practice is unfair to the public, to the profession as a whole, and to radiology. On such occasions it is, however, more the duty of the radiologists in the city or cities in question to try to cope with such a state of affairs than it is for the College to attempt a solution of the problem. No radiologist should have a connection of this type with any hospital and I believe in time

[1] Delivered before the American College of Radiology, New York City, June 12, 1940.

all fair men will discontinue such phlegmatic service. Certainly men in other specialties are not going to respect a radiologic department operated in this manner. It also breeds lack of respect for radiology among interns. In a city where this type of work is done, it will almost invariably be found that radiology is not looked upon by the profession as being on a par with, or equal to, the other specialties. In such cities rarely does one find a radiologist in an official capacity in his local medical society. In other words, rarely is he respected as a real consultant. If one or more men give very poor service to their hospital, it also reflects discredit among their colleagues in radiology in their communities. If they should have any trouble with a hospital, the profession as a whole will not stand behind them or back them in their fight with some grasping hospital board or medical group. Co-operation among radiologists in any city is absolutely essential if radiology is to rate as high as any of the other specialties in medicine. In cases in which good radiology is done and in which the radiologists co-operate with each other and are deeply interested in helping the clinician in every case referred to him, this specialty stands just as well as any other in medicine.

I would like to condemn strongly the habit of some radiologists in remaining in their private offices and sending technicians to the hospitals for x-ray examination of patients. In this way the importance of the technical side is exaggerated by the staff and the interpretation of the films minimized. Nor does he come in personal contact with the clinician or the patient and make friends of them.

If an established radiologist has more hospital work than he can thoroughly attend to, he should have well trained associates who can help him give proper service to these hospitals. He cannot be too grasping and fail to bring discredit upon his specialty.

If he is giving good service to the hospital and has made friends of the staff members, he is more apt to be able to prevent the inclusion of radiology in the various plans for hospitalization. We should, I believe, be unalterably opposed to radiology's being included in hospital plans unless the other specialties are also included. Various forms of low cost medical service to low income groups are already operating satisfactorily in several states. Some physicians are opposed to this medical service but those who have had more experience with it feel that it is a good thing and that the radiologist should do his share in helping complete such plans in various parts of the country.

Two months ago I was in the office of one of America's leading radiologists and asked him what would be his subject if he had to make this talk. His immediate reply was, "If radiology is to succeed, radiologists must be consultants." He also added, "They must not only be interested in radiology itself, but must be thoroughly interested in trying to give the best possible service to the public, the medical profession as a whole, the hospital, and to radiology. He must willingly absorb the cost of a good deal of charity work and must feel that it is up to him to give the referring physician as much help as he possibly can."

More than 90 per cent of radiologists must deal with a large preponderance of general practitioners. The referring physician wants a diagnosis and in many instances suggestions as to treatment. Many men in our field have hundreds of doctors who may send in a patient with a note which reads something like this: "This patient has a lesion on the face, tongue, etc. Please treat it as you think you should. If you do not think it is a case for you to handle, please call me and advise me as to what treatment you think is indicated." A radiologist who has this type of confidence of the profession as a whole will always succeed. He naturally must be extremely careful not to abuse this confidence.

The reason pathology has suffered so much at the hands of hospitals and medical groups is due largely to the fact that the pathologist has little personal contact with the patient. It is, of course, realized that in very large clinics it is impossible for the radiologist to see many patients personally. He has, however, daily consultations with members of the staff and in this way is familiarized with the patient as a whole and not with just one organ or part of the body. In smaller institutions and in private practice, the radiologist in charge of the department or one of his associates should see every patient, and should try in every possible way not only to help the clinician who referred the case, but to make a friend of the patient. All too frequently the patient has no idea of the name of the radiologist or anything else concerning him. Many, many times I have asked the question, "Who x-rayed you?" The answer in many cases has been, "I do not know." "Dr. So and So sent me for the examination." In a certain percentage of cases they not only know by whom they have

been x-rayed but a good deal about the radiologist who did the work. I am probably old fashioned, but I believe that a radiologist should have patients of his own, particularly in therapy. Such men as Groover felt that if radiology was to remain on a par with other specialties, the radiologist must refer patients as well as have patients referred to him. There is no reason why this should not be true in many dermatologic conditions, in cancer, particularly skin cancer, etc.

To-day, nearly all specialists start the study of their chosen field after one or two years of hospital internship. They work from around 9:00 A.M. to 5:00 P.M. and feel that the day is over. They have several years of this without much night or Sunday work. It is true that many of the younger men in the various specialties can operate, read films, etc., as well as older men but some of these do not wish to be disturbed after 5:00 P.M. or on Sundays and have no real conception of service to the public, the medical profession, or to their own specialty. They have never done general practice in which the phone may ring any hour of the day or night and service must be cheerfully rendered if they can possibly hope to build a practice or make a living for themselves.

Radiologists must attend staff meetings and be sincerely interested in trying to understand and help other physicians with their troubles if they are to expect their specialty to stand on a par with the others. The College of Radiology has, I am firmly convinced, helped the general standing of radiologists throughout the country and is helping them in their hospital relationship.

Just what can we do to help the College and the radiologic societies in advancing the position of radiology?

First, we can work together in a spirit of cooperation. If a radiologist is giving good service to a hospital or medical group, he should be able to feel that other reputable radiologists are not going to undermine or underbid him in his work. For example: In one city a radiologist resigned from a hospital on account of the fact that he was being paid only about $200 a month for an enormous amount of work. The other radiologists in this city immediately met and decided that none of them would consider the appointment to this hospital. The final outcome was that the radiologist's salary was raised sufficiently for him to continue the work. His success would not have been possible except for the co-operation of all of the radiologists in his community. Some will say this is using union tactics. It is not at all. No other branch of medicine would even consider allowing itself to be so commercialized or exploited. On the other hand, in another city a radiologist is giving extremely poor service to one of the hospitals. The other men in this community feel that his actions are lowering the standard of radiology and want the College to do something about it. They are, however, unwilling to come out in the open against this man. Naturally, the College can do nothing under such circumstances.

There is no question whatever that in the large majority of instances in all of our dealings with hospitals and the profession, education, service, and moral suasion are far better than any method which smacks of force or union tactics. Samuel Gompers said, "One fact stands out in bold relief in the history of men's attempts for betterment. That is when compulsion is used, only resentment is aroused, and the end is not gained. Only through moral suasion and appeal to men's reason can a movement succeed." This does not mean, however, that in certain instances we should not, when driven to it, fight for what is right and proper.

Often the radiologist is not giving service to the hospital but complains bitterly because some younger man who will give service is offered his position. At times hospital boards are absolutely unreasonable. A radiologist, though giving excellent service, may have trouble. Other doctors have their troubles. We must expect ours. Carlyle said, "No man lives without jostling and being jostled; in all ways he has to elbow himself through the world, giving and receiving offence."

Another thing that we can do to help the College in advancing its objectives is for all of us to try to interest men of high type to enter this field. We cannot expect a radiologist of small caliber to compete with surgeons, internists, and others of much higher type. It is true that at the present time not enough men are being trained in this field to take care fully of the specialty. Every town of 10,000 persons with a good surrounding country could well support a good radiologist. It seems to be true that a good many men are going to have to be partially trained in smaller cities throughout the country and complete their courses in special work in some of the larger centers.

Unless some arrangement of this kind is made, we are obliged to expect poor radiology in many parts of America.

Since radiologists at one time considered their offices laboratories, they have in many instances failed to do all that they could for their specialty and by remaining more or less secluded have, though doing excellent work, been more or less unknown to the profession as a whole and to the laymen in their own communities. They should take an active part in their local medical society and in the national medical societies.

Through papers published and through our excellent radiologic journals, our specialty is undoubtedly advancing in the scientific world and radiologists are now more generally known by the profession than has been the case in the past. As much as Dr. Pancoast did for radiology, a few years ago a professor of surgery in one of the largest universities in the East, and not a great distance from Philadelphia, on hearing Dr. Pancoast's name mentioned, asked, "Who is Dr. Pancoast?"

In the future, I believe that in the larger cities radiologists should, where possible, form groups in which special study can be given to the different fields of radiology. This enables the group to have better equipment and to be more familiar with all of the different branches of radiology than is possible in smaller offices. It lowers the overhead and such a group is able to turn out better work and can more easily compete with the clinician who attempts to do his own radiology. I believe that we are obliged to have more or less specialists within the specialty if the best possible type of work is to be turned out. The large majority of unusually successful men in all branches of medicine have been particularly interested in some one phase of their specialty and have become more proficient in this more or less specialty within a specialty.

One of the things which would be an experiment but which the College could probably do to help radiologists throughout the country would be to form a department for handling a number of transparencies and case histories in some of the highly specialized fields, these to be available to any radiologist who desired them and was willing to pay for them. For instance: A number of transparencies with case histories and final diagnoses in head conditions by Dyke; a similar series on the small intestine by Golden; kymography and planigraphy by Moore and Scott, etc., would be a tremendous help to radiologists throughout the country. I would recommend that the College take steps to determine the number of its membership which would desire this service and attempt some approximation of the expense which would be incurred.

Most of the work in the College has been done by a few public spirited men who have given a great deal of time and effort to the College. All of us are very much indebted to the entire board of chancellors for the work done during 1939 and 1940. Special thanks are due Dr. Edward Chamberlain, chairman of the board; to Dr. Ross Golden, chairman of the committee on constitution and by-laws; and to our executive secretary, Mr. Cahal.

The road to success in radiology is not a simple one but I predict a great future for this specialty if we can follow Mark Twain's advice, "Let us endeavor so to live that when we come to die even the undertaker will be sorry."

Richmond, Va.

FRED M. HODGES, M.D.

RADIOLOGICAL SOCIETIES IN NORTH AMERICA

Editor's Note.—Will secretaries of societies please cooperate with the Editor by supplying him with information for this section? Please send such information to Leon J. Menville, M.D., 1201 Maison Blanche Bldg., New Orleans, La.

UNITED STATES

CALIFORNIA

California Medical Association, Section on Radiology.—Chairman, Karl M. Bonoff, M.D., 1930 Wilshire Blvd., Los Angeles; *Secretary,* Carl D. Benninghoven, M.D., 95 S. El Camino Real, San Mateo.

Los Angeles County Medical Association, Radiological Section.—President, M. L. Pindell, M.D.; *Vice-president,* Richard T. Taylor, M.D.; *Secretary,* Wilbur Bailey, M.D., 2007 Wilshire Blvd.; *Treasurer,* Henry Snure, M.D., 1414 South Hope Street; Kenneth Davis, M.D., *Member of Executive Committee.* Meets second Wednesday of each month at County Society Building.

Pacific Roentgen Society.—Chairman, William E. Costolow, M.D., Los Angeles; *Members of Executive Committee,* I. S. Ingber, M.D., San Francisco; D. R. MacColl, M.D., Los Angeles, and J. D. Coate, M.D., Oakland; *Secretary-Treasurer,* L. Henry Garland, M.D., 450 Sutter St., San Francisco. Executive Committee meets quarterly; Society meets annually during annual meeting of the California Medical Association.

San Francisco Radiological Society.—Secretary, Harold A. Hill, M.D., 450 Sutter Street. Meets monthly on third Thursday at 7:45 P.M., for the first six months at Toland Hall (Univ. of Calif. Med. School) and for the second six months at Lane Hall (Stanford Univ. School of Med.).

COLORADO

Denver Radiological Club.—President, N. B. Newcomer, M.D., 306 Republic Bldg.; *Vice-president,* Elizabeth Newcomer, M.D.; *Secretary,* Paul R. Weeks, M.D., 520 Republic Bldg.; *Treasurer,* L. G. Crosby, M.D., 366 Metropolitan Bldg. Meets third Friday of each month at homes of members.

CONNECTICUT

Connecticut State Medical Society, Section on Radiology.—Chairman, Owen J. Groark, M.D., 881 Lafayette St., Bridgeport; *Secretary-Treasurer,* Max Climan, M.D., 242 Trumbull St., Hartford. Meetings twice annually in May and September.

DELAWARE

Affiliated with Philadelphia Roentgen Ray Society.

FLORIDA

Florida Radiological Society.—President, J. H. Lucinian, M.D.; *Vice-president,* John N. Moore, M.D.; *Secretary-Treasurer,* Elliott M. Hendricks, M.D., 314 Sweet Bldg., Fort Lauderdale. The Fall Meeting will be held Nov. 2 and 3, 1940, at the Miami Biltmore Hotel, and at the annual meeting of the Medical Association of Florida in the spring.

GEORGIA

Georgia Radiological Society.—President, Robert Drane, M.D., DeRenne Apts., Savannah; *Vice-president,* J. J. Collins, M.D., Archbold Hospital, Thomasville; *Secretary-Treasurer,* Robert C. Pendergrass, M.D., Prather Clinic Bldg., Americus. Meetings twice annually, in November and at the annual meeting of the Medical Association of Georgia in the spring.

ILLINOIS

Chicago Roentgen Society.—President, Adolph Hartung, M.D.; *Vice-president,* Warren W. Furey, M.D.; *Secretary,* Chester J. Challenger, M.D., 3117 Logan Blvd. The Society meets at the Palmer House on the second Thursday of October, November, January, February, March, and April.

Illinois Radiological Society.—President, Harry W. Ackeman, M.D., 321 W. State St., Rockford; *Vice-president,* D. R. Hanley, M.D., St. Mary's Hospital, Streator; *Secretary-Treasurer,* William DeHollander, M.D., St. John's Hospital, Springfield. Meetings quarterly by announcement.

Illinois State Medical Society, Section on Radiology.—Chairman, Warren W. Furey, M.D., 6844 Oglesby Ave., Chicago; *Secretary,* Harry W. Ackeman, M.D., 321 W. State St., Rockford.

INDIANA

The Indiana Roentgen Society.—President, H. H. Inlow, M.D., Shelbyville; *President-elect,* Charles Wyeth, M.D., Terre Haute; *Vice-president,* C. A. Stayton, M.D., Indianapolis; *Secretary-Treasurer,* Clifford C. Taylor, M.D., 23 E. Ohio St., Indianapolis. Annual meeting in May.

IOWA

The Iowa X-ray Club.—Holds luncheon and business meeting during annual session of Iowa State Medical Society.

KENTUCKY

Kentucky Radiological Society.—President, D. B. Harding, M.D., Lexington; *Vice-president,* I. T. Fugate, M.D., Louisville; *Secretary-Treasurer,* Joseph C. Bell, M.D., 402 Heyburn Bldg., Louisville. Meeting annually in Louisville, third Sunday afternoon in April.

MAINE

See New England Roentgen Ray Society.

MARYLAND

Baltimore City Medical Society, Radiological Section.—Chairman, Harold E. Wright, M.D., 101 W. Read St.; *Secretary,* Walter L. Kilby, M.D., 101 W. Read St. Meetings are held the third Tuesday of each month.

MASSACHUSETTS

See New England Roentgen Ray Society.

MICHIGAN

Detroit X-ray and Radium Society.—President, O. J. Shore, M.D., 552 Fisher Bldg., Detroit; *Vice-president,* Clarence E. Hufford, M.D., 421 Michigan St., Toledo, Ohio; *Secretary-Treasurer,* E. R. Witwer, M.D., Harper Hospital, Detroit. Meetings first Thursday of each month from October to May, inclusive, at Wayne County Medical Society club rooms, 4421 Woodward Ave., Detroit.

Michigan Association of Roentgenologists.—President, J. H. Dempster, M.D., Detroit; *Vice-president,* L. E. Holly, M.D., Muskegon; *Secretary-Treasurer,* J. E. Lofstrom, M.D., 1536 David Whitney Bldg., Detroit. Meetings quarterly by announcement.

MINNESOTA

Minnesota Radiological Society.—President, Harry Weber, M.D., Mayo Clinic, Rochester; *Vice-president,* G. T. Nordin, M.D., Minneapolis; *Secretary,* John P. Medelman, M.D., 572 Lowry Medical Arts Bldg., St. Paul. Meetings quarterly.

MISSOURI

The Kansas City Radiological Society.—President, L. G. Allen, M.D., 907 N. 7th St., Kansas City, Kansas; *Secretary,* Ira H. Lockwood, M.D., 306 E. 12th St., Kansas City, Mo. Meetings last Thursday of each month.

The St. Louis Society of Radiologists.—President, Oscar C. Zink, M.D., St. Luke's Hospital; *Secretary,* Wilbur K. Mueller, M.D., University Club Bldg. Meets on fourth Wednesday of October, January, March, and May, at a place designated by the president.

NEBRASKA

Nebraska Radiological Society.—President, Roy W. Fouts, M.D., 1007 Medical Arts Bldg., Omaha; *Secretary,* D. Arnold Dowell, M.D., 816 Medical Arts Bldg., Omaha. Meetings third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY

(Maine, New Hampshire, Vermont, Massachusetts, and Rhode Island.) *Secretary,* Hugh F. Hare, M.D., Lahey Clinic, Boston, Mass. Meets monthly on third Friday at Boston Medical Library.

NEW HAMPSHIRE

See New England Roentgen Ray Society.

NEW JERSEY

Radiological Society of New Jersey.—President, James G. Boyes, M.D., 912 Prospect Ave., Plainfield; *Vice-president,* Nathan J. Furst, M.D., 190 Johnson Ave., Newark; *Secretary,* W. James Marquis, M.D., 198 Clinton Ave., Newark; *Treasurer,* H. A. Vogel, M.D., 1060 East Jersey St., Elizabeth, and *Counsellor,* H. J. Perlberg, M.D., 921 Bergen Ave., Jersey City. Meetings at Atlantic City at time of State Medical Society, and Midwinter in Newark as called by president.

NEW YORK

Associated Radiologists of New York, Inc.—President, I. J. Landsman, M.D., 910 Grand Concourse, New York City; *President-elect,* D. E. Ehrlich, M.D., 35 West 92nd St., New York City; *Vice-president,* Frederic E. Elliott, M.D., 122 76th St., Brooklyn; *Treasurer,* Solomon Fineman, M.D., 133 East 58th St., New York City; *Secretary,* William J. Francis, M.D., 210 Fifth Ave., New York City. Regular meetings the first Monday evening of the month in March, May, October, and December.

Brooklyn Roentgen Ray Society.—President, A. L. L. Bell, M.D., Long Island College Hospital, Henry, Pacific, and Amity Sts.; *Secretary-Treasurer,* L. J. Taormina, M.D., 1093 Gates Ave. Meetings first Tuesday in each month at place designated by president.

Buffalo Radiological Society.—President, Edward Koenig, M.D., 100 High St., Buffalo; *Vice-president,* W. Roger Scott, M.D., 598 Pine St., Niagara Falls; *Secretary-Treasurer,* Joseph S. Gian-Franceschi, M.D., 610 Niagara St. Meetings second Monday evening each month, October to May, inclusive.

Central New York Roentgen Ray Society.—President, Albert Lenz, M.D., 613 State St., Schenectady; *Vice-president,* Dwight V. Needham, M.D., 123 Sedgwick St., Syracuse; *Secretary-Treasurer,* Carlton F. Potter, M.D., 425 Waverly Ave., Syracuse. Meetings are held in January, May, and October, as called by Executive Committee.

Long Island Radiological Society.—President, Samuel G. Schenck, M.D., Brooklyn; *Vice-president,* G. Henry Koiransky, M.D., Long Island City; *Secretary,* Marcus Wiener, M.D., 1480 48th St., Brooklyn; *Treasurer,* Louis Goldfarb, M.D., 608 Ocean Ave., Brooklyn. Meetings fourth Thursday evening each month at Kings County Medical Bldg.

New York Roentgen Society.—President, Henry K. Taylor, M.D., 667 Madison Ave., New York City;

Vice-president, Roy D. Duckworth, M.D., 170 Maple Ave., White Plains, N. Y.; *Secretary*, Eric J. Ryan, M.D., St. Luke's Hospital, New York City, and *Treasurer*, Paul C. Swenson, M.D., 168th St. and Broadway, New York City.

Rochester Roentgen-ray Society.—Chairman, George H. S. Ramsey, M.D., 277 Alexander St.; *Secretary*, S. C. Davidson, M.D., 277 Alexander St. Meetings at convenience of committee.

NORTH CAROLINA

Radiological Society of North Carolina.—President, Robert P. Noble, M.D., 127 W. Hargett St., Raleigh; *Vice-president*, A. L. Daughtridge, M.D., 144 Coast Line St., Rocky Mount; *Secretary-Treasurer*, Major I. Fleming, M.D., 404 Falls Road, Rocky Mount. Meetings with State meeting in May, and meeting in October.

OHIO

Ohio Radiological Society.—President, U. V. Portmann, M.D., Cleveland; *Secretary*, J. E. McCarthy, M.D., Cincinnati. A committee was appointed to draw up a constitution and by-laws. The next meeting will be held at the time and place of the annual meeting of the Ohio State Medical Association.

Cleveland Radiological Society.—President, L. A. Pomeroy, M.D., Hanna Bldg., Cleveland; *Vice-president*, P. C. Langan, M.D., 215 Wellsley Ave., Akron; *Secretary-Treasurer*, H. A. Mahrer, M.D., 10515 Carnegie Ave., Cleveland. Meetings at 6:30 P.M. at the Mid-day Club, in the Union Commerce Bldg., on fourth Monday of each month from October to April, inclusive.

Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*President*, Samuel Brown, M.D.; *Secretary-Treasurer*, Justin E. McCarthy, M.D., 707 Race St. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—President, H. Norton Mawhinney, M.D., Pittsburgh; *President-elect*, Peter B. Mulligan, M.D., Ashland; *First Vice-president*, Harold S. Callen, M.D., Bradford; *Second Vice-president*, Harold W. Jacox, M.D., Pittsburgh; *Secretary-Treasurer*, L. E. Wurster, M.D., 416 Pine St., Williamsport; *Editor*, William E. Reiley, M.D., Clearfield; *Assistant Editor*, Sydney J. Hawley, M.D., Danville; *Censor for Three Years*, A. R. Snedden, M.D., McKeesport. The Society meets annually; time and place of next meeting will be announced later.

The Philadelphia Roentgen Ray Society.—President, Jacob H. Vastine, II, M.D., Medical Arts Bldg., Philadelphia; *Vice-president*, A. Maxwell Sharpe, M.D., 708 Sproul St., Chester; *Secretary*, Barton R. Young, M.D., Temple University Hospital, Philadelphia; *Treasurer*, Fay K. Alexander, M.D., Chestnut Hill Hospital, Philadelphia. Meetings held first Thursday of each month at 8:15 P.M., from October to May, in Thomson Hall, College of Physicians, 21 S. 22nd St., Philadelphia.

The Pittsburgh Roentgen Society.—President, Paul G. Bovard, M.D., 306 Corbett St., Tarentum, Pa.; *Vice-president*, John H. Gemmell, M.D., 262 Connecticut Ave., Rochester, Pa., and *Secretary-Treasurer*, Harold W. Jacox, M.D., 4800 Friendship Ave., Pittsburgh, Pa. Meetings held second Wednesday of each month at 4:30 P.M., from October to June, at various hospitals designated by program committee.

RHODE ISLAND

See New England Roentgen Ray Society.

SOUTH CAROLINA

South Carolina X-ray Society.—President, T. A. Pitts, M.D., Columbia; *Secretary-Treasurer*, Malcolm Mosteller, M.D., Columbia Hospital, Columbia. Meetings in Charleston on first Thursday in November, also at time and place of South Carolina State Medical Association.

SOUTH DAKOTA

Meets with Minnesota Radiological Society.

TENNESSEE

Memphis Roentgen Club.—Chairmanship rotates monthly in alphabetical order. Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—President, Eugene Abercrombie, M.D., 305 Medical Arts Bldg., Knoxville; *Vice-president*, Christopher C. McClure, M.D., 404 Doctors Bldg., Nashville; *Secretary-Treasurer*, Franklin B. Bogart, M.D., 311 Medical Bldg., Chattanooga. Meeting annually with State Medical Society in April.

TEXAS

Texas Radiological Society.—President, C. F. Crain, M.D., Corpus Christi; *President-elect*, M. H. Glover, M.D., Wichita Falls; *First Vice-president*, G. D. Carlson, M.D., Dallas; *Second Vice-president*, P. E. Wigby, M.D., Dallas; *Secretary-Treasurer*, L. W. Baird, M.D., Scott and White Hospital, Temple. Meets annually. The next annual meeting is to be Jan. 18, 1941, in Sherman.

VERMONT

See New England Roentgen Ray Society.

VIRGINIA

Virginia Radiological Society.—President, Wright Clarkson, M.D., Petersburg; *Vice-president*, Clayton

W. Ely, M.D., Norfolk; *Secretary*, Charles H. Peterson, M.D., 603 Medical Arts Bldg., Roanoke.

The annual meeting of the Virginia Radiological Society was held at the Greenbrier Hotel, White Sulphur Springs, West Virginia, on July 29, 1940. This society meets annually in October, but the meeting this year was held earlier in order to coincide with the joint meeting of the Virginia and West Virginia Medical Societies.

WASHINGTON

Washington State Radiological Society.—President, H. E. Nichols, M.D., Stimson Bldg., Seattle; *Vice-president*, George Cornett, M.D., Yakima; *Secretary-Treasurer*, Kenneth J. Holtz, M.D., American Bank Bldg., Seattle. Meetings fourth Monday of each month at College Club, Seattle.

WISCONSIN

Milwaukee Roentgen Ray Society.—President, H. W. Hefke, M.D.; *Vice-president*, Frederick C. Christensen, M.D.; *Secretary-Treasurer*, Irving I. Cowan, M.D., Mount Sinai Hospital, Milwaukee. Meets monthly on first Friday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, Russel F. Wilson, M.D., Beloit Municipal Hospital, Beloit. Two-day annual meeting in May and one day in connection with annual meeting of State Medical Society, in September.

University of Wisconsin Radiological Conference.—Secretary, E. A. Pohle, M.D., 1300 University Ave., Madison, Wis. Meets every Thursday from 4 to 5 P.M., Room 301, Service Memorial Institute.

CANADA

Section on Radiology, Canadian Medical Association.—Chairman, Gordon Richards, M.D., Medical Arts Bldg., Toronto; *Secretary*, W. J. Cryderman, M.D., Medical Arts Bldg., Toronto.

Section on Radiology, Ontario Medical Association.—Chairman, E. H. Shannon, M.D., St. Michael's Hospital, Toronto; *Secretary*, W. J. Cryderman, M.D., 474 Glenlake Avenue, Toronto.

Canadian Association of Radiologists.—President, J. E. Gendreau, M.D., Montreal; *Vice-president*, W. H. McGuffin, M.D., Calgary; *Honorary Secretary-Treasurer*, W. L. Ritchie, M.D., Montreal; *Chairman of Interrelations Committee*, G. E. Richards, M.D., Toronto.

EDITORIAL

LEON J. MENVILLE, M.D., *Editor* HOWARD P. DOUB, M.D., *Associate Editor*

THE AMERICAN SOCIETY OF X-RAY TECHNICIANS

The intimate association of radiologists and their technical assistants and the dependence of the one on the other is so well recognized that it does not require comment. However, the organization which the technicians have set up has had rather scant recognition by radiologists, as a whole. This may be due to the fact that many of them resented the desire on the part of their employees to organize. The fear of some that such an organization might become the prey of organized labor has not materialized.

Fortunately, there have always been a few radiologists who have been willing to give freely of their time and advice to the technicians. This Society has long had a standing committee whose function has been to promote radiology insofar as it concerned the relationship of the radiologist and the technician. Under their guidance the American Society of X-ray Technicians has been organized along strictly ethical lines. The only aim has been the professional improvement of their members and the elevation of the standards of their work. A registry of technicians has been established that certifies those who, after examination, are found to be properly qualified, in much the same manner as does our American Board of Radiology.

Any group as large as the technicians of the United States and Canada will sooner or later feel the need of a national organization. It is better for us, as radiologists, to support the present one which has been sponsored by prominent members of our specialty than to encourage the formation of another society that does not desire any advice or guidance from us. The code of ethics of the American Society of X-ray Technicians precludes any encroachment by them upon our field. Neither does it prevent the radiologist from regulating the technic in his office or department in any way he desires.

The criticisms of the American Society of X-ray Technicians were well answered in an editorial by Ernst A. Pohle, M.D., in the October, 1939, issue of RADIOLOGY. The present national society, with its code of ethics, will do more than any other single agency to prevent the divorcement of the medical and technical sides of radiology.

C. H. HEACOCK, M.D.

Memphis, Tenn.

THE NEXT ANNUAL MEETING

AN INVITATION TO THE ANNUAL MEETING OF THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

It is with great pleasure that, as President of the Radiological Society of North America, I extend this invitation to the members of the Society and to the medical profession as a whole to attend the Annual Meeting of the Society to be held in Cleveland from December the second to the sixth, at the Statler Hotel.

The meeting this year will offer most excellent Refresher Courses, beginning on Sunday, December the first, and continuing for a two-hour period each morning during the meeting. It will be necessary for members to obtain tickets for these sessions. The response for the Scientific Section presages a splendid Scientific Exhibit. The Commercial Exhibit has reserved a huge space where all the latest instruments and devices will be demonstrated by courteous commercial exhibitors.

The Entertainment Committee has made elaborate social plans for the meeting. On Wednesday night, in particular, a large downtown dinner club has been taken over, and the members are invited to bring their wives and friends for a gala evening with dinner, entertainment, and dancing. This will take the place of the usual stag night. A small charge will be made for this evening and an excellent time should be had by all. The commercial exhibitors are cordially invited to participate in this night of entertainment.

There has been a most hearty response from the members and invited speakers for this program, for what we believe will be a most instructive meeting. Make your reservations early. Come, see, hear, and make merry!

BERNARD H. NICHOLS, M.D.
President

PRELIMINARY PROGRAM

The Twenty-sixth Annual Meeting of the Radiological Society of North America will be held in Cleveland, at the Hotel Statler, Dec. 2–6, 1940.

That the Technical Exhibit is going to be noteworthy is indicated by the unprecedented demand for space by exhibitors of the highest type. This Exhibit gives promise of being well worth the study of radiologists.

The Scientific Exhibit is being collected under the Chairmanship of E. R. Witwer, M.D., of the Harper Hospital, Detroit, and will undoubtedly be of our usual high standard of excellence and well worth consideration and study.

Announcement of the Refresher Series of Post-graduate Courses, in detail, was made in RADIOLOGY for September, together with an Enrollment Blank. All those planning to attend the Annual Meeting will read with interest the schedule of courses offered.

There follows an enumeration of papers to be presented at the Scientific Sessions, though not necessarily in the order here given. Under the able leadership of our President, Bernard H. Nichols, M.D., of Cleveland, the Society anticipates a remarkable scientific session.

"Standard Nomenclature for Lesions in the Vertebræ." ARIAL W. GEORGE, M.D., of Boston.

"Soft Tissue Radiography." JOHN R. CARTY, M.D., of New York City.

"Gastro-colic Fistula." MAX RITVO, M.D., of Boston.

"Intrathoracic Neurofibromas." KARL KORNBLUM, M.D., of Philadelphia.

"A Study of the Intestinal Tract of Children and its Relation to the Adult." W. WALTER WASSON, M.D., of Denver.

"Compression of the Heart." CLAUDE S. BECK, M.D., of Cleveland.

"Renal Tuberculosis." GILBERT THOMAS, M.D., of Minneapolis.

"Roentgenologic Consideration of the Normal Mucosa of the Duodenum." LEO G. RIGLER, M.D., of Minneapolis.

"Silicosis." GAGE CLEMENT, M.D., of Duluth, Minn.

"The Bone Changes in Idiopathic Steatorrhea." HUGH F. HARE, M.D., of Boston.

"Pyelography (Done in the Upright Position)." J. ANDREW BOWEN, M.D., of Louisville, Ky.

"Pancreatic Tumors." SAMUEL BROWN, M.D., of Cincinnati, Ohio.

"Mediastinal and Pulmonary Changes in Erythema Nodosum." ERNST A. POHLE, M.D., and LESTER W. PAUL, M.D., of Madison, Wisc.

"Congenital Cysts of the Lung." LAWRENCE REYNOLDS, M.D., of Detroit.

"Intravenous Urography for Children." GEORGE M. WYATT, M.D., of Boston.

"Nasal Accessory Sinuses." GEORGE W. GRIER, M.D., of Pittsburgh, Pa.

"Ventriculography and Diagnosis of Brain Tumors." MAX M. PEET, M.D., of Ann Arbor, Mich.

"Measurement of Visual Acuity by Roentgen Ray." R. R. NEWELL, M.D., of San Francisco.

"Pseudo-fractures in Diseases Affecting the Skeletal System." JOHN D. CAMP, M.D., of Rochester, Minn.

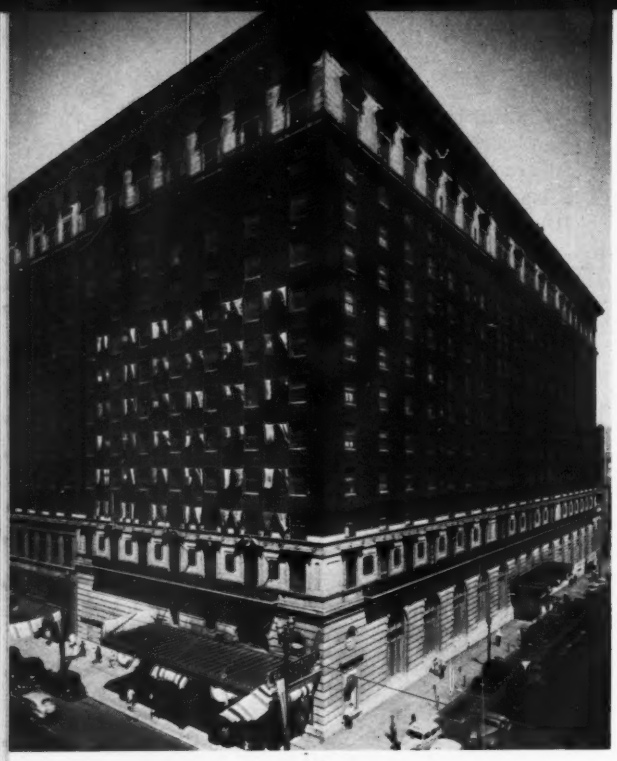
"Alkaptonuria, Ochronosis, and Arthritis." MAURICE M. POMERANZ, M.D., of New York City.

Tumor Clinic. Lakeside Hospital, Cleveland.

"Cholangiography, Fractional Method." W. S. ALTMAN, M.D., of Quincy, Mass.

"Accuracy of Focal Spot Size Determination." LEWIS G. JACOBS, M.D., of Winona, Minn.

"The Criteria for the Diagnosis of Malignancy." ALLEN GRAHAM, M.D., of Cleveland.



Exterior View, Hotel Statler, Cleveland.

"Present Status of X-ray Therapy in Cancer of the Breast." MAURICE LENZ, M.D., of New York City.

"Giant-cell Tumors of Bone." E. R. WITWER, M.D., and T. LEUCUTIA, M.D., of Detroit.

"Roentgen Irradiation on Chronic Lymphatic Leukemia." W. C. POPP, M.D., and C. H. WATKINS, M.D., of Rochester, Minn.

"Depth Dose Measurements by Means of Goldfish." F. P. ELLINGER, M.D., of New York City, and R. CROSS, M.D., of Kansas City, Mo.

"Distribution of Ionizing Energy in the Human Body Produced by Different Methods of Treatment." PAUL AEBERSOLD, Ph.D., of San Francisco.

"Irradiation Methods in Carcinoma of the Cervix Uteri in Actual Practice." ERIC LILJENCRANTZ, M.D., of San Francisco.

"Roentgen Irradiation of the Pelvis in Carcinoma of the Cervix Uteri: Our Present Method." R. S. STONE, M.D., and J. M. ROBINSON, M.D., of San Francisco.

"Irradiation in Carcinoma of the Cervix Uteri." S. T. CANTRIL, M.D., and F. J. BUSCHKE, M.D., of Chicago.

"Irradiation Changes in Germ Cells." P. S. HENSHAW, Ph.D., of Bethesda, Md.

"The Elimination of Thorotrast." K. W. STENSTROM, Ph.D., of Minneapolis.

"Studies on the Concentration of Radiophosphorus in Normal and Disease Lymph Nodes." J. M. KENNY, M.D., and L. D. MARINELLI, M.A., of New York City.

"Studies on the Concentration of Organic Dyes in Tumor Tissue (in Connection with Artificial Radio-activity)." DR. ZAHL.

"The Absorption of Radio-phosphorus in Irradiated and Non-irradiated Mice." L. D. MARINELLI, M.A., and J. M. KENNY, M.D., of New York City.

"Biological Applications of Micro-radiography." G. L. CLARK, Ph.D., of Urbana, Ill.

"The New High Voltage X-ray Standardization Laboratory of the N.B.S." LAURISTON S. TAYLOR, Ph.D., of Washington, D. C.

"Recent Contribution of Physics and Engineering Research to Radiology." B. CASSEN, Ph.D., of Pittsburgh, Pa.

"The Development of Dosimetry in X-ray Therapy." O. GLASSER, Ph.D., of Cleveland.

"The Effect of Shock-proof Cables and Condensers on Two-valve, Half-wave Rectification." M. M. D. WILLIAMS, Ph.D., of Rochester, Minn.

"Irradiation in Dermatology." A. CIPOLLARO, M.D., of New York City.

"The Treatment of Hemangiomas in Children." F. M. HODGES, M.D., and R. A. BERGER, M.D., of Richmond, Va.

"The Roentgen Treatment of Myxomatous Cutaneous Cysts." H. W. JACOX, M.D., and DR. FREEDMAN of Pittsburgh, Pa.

"Roentgen Therapy of Tuberculous Cervical Lymphadenitis." H. HAUSER, M.D., of Cleveland.

"Irradiation in the Treatment of Carcinoma of the Rectum." H. H. BOWING, M.D., of Rochester, Minn.

"Combined Irradiation and Surgery in the Treatment of Carcinoma of the Pelvic Colon." HOWARD P. DOUB, M.D., J. P. PRATT, M.D., and H. C. JONES, M.D., of Detroit.

"Contact Irradiation in Carcinoma of the Bladder." L. S. GOIN, M.D., of Los Angeles.

"Serial Biopsy in the Irradiation Treatment of Tumors of the Upper Respiratory Tract." M. FRIEDMAN, M.D., and J. W. HALL, M.D., of New York City.

"Carcinoma of the Male Breast: A Review of 200 Cases." M. D. SACHS, M.D., of Portland, Oregon.

"The Treatment of Intra-oral Carcinoma: A Review of 110 Cases." W. E. HOWES, M.D., and DR. BERNSTEIN, of Brooklyn, N. Y.

"Roentgen Irradiation of Calcareous Deposits about the Shoulder." L. W. BAIRD, M.D., of Temple, Texas.

"The Effect of Roentgen Therapy on Closure of the Epiphyses." DAVIS SPANGLER, M.D., of Dallas, Texas.

"Irradiation Therapy of Brain Tumors." IRA I. KAPLAN, M.D., of New York City.

COMMUNICATIONS

MIDSUMMER RADIOLOGICAL CONFERENCE

The Sixth Midsummer Radiological Conference in the Rocky Mountains was held in Denver, Colo., Aug. 8, 9, and 10, 1940, with headquarters at the Hotel Shirley-Savoy. The meeting was sponsored by the Denver Radiological Club under the presidency of Nathan B. Newcomer, M.D. There was a total registration of 250 from 15 States.

A noticeable feature of the meeting was the loyal co-operation of the local medical profession, demonstrating what can be accomplished by a small group of radiologists when such co-operation is secured.

At the banquet on Friday evening, Albert Soiland, M.D., of Los Angeles, and James M. Martin, M.D., of Dallas (through his son, Charles Martin, M.D.), were presented silver plaques in recognition of their distinguished services to radiology.

The following guest speakers participated in the scientific sessions: Lewis G. Allen, M.D., of Kansas City, Kans.; Fred E. Angle, M.D., of Kansas City, Kans.; Howard P. Doub, M.D., of Detroit, Mich.; Edwin C. Ernst, M.D., of St. Louis, Mo.; Howard B. Hunt, M.D., of Omaha, Nebr.; Charles L. Martin, M.D., of Dallas, Texas, and Albert Soiland, M.D., of Los Angeles, Calif.

In addition to the guest speakers, papers were read by Charles G. Freed, M.D., of Denver; J. Rudolph Jaeger, M.D., of Denver; William R. Lipscomb, M.D., of Denver; Ralph M. Stuck, M.D., of Denver; Kenneth D. A. Allen, M.D., of Denver; Lloyd R. Allen, M.D., of Colorado Springs; Edward J. Meister, M.D., and John W. McMullen, M.D., of Denver, and W. Walter Wasson, M.D., of Denver.

There was also a very excellent scientific exhibit.

Terrace Dining Room, Hotel Statler, Cleveland.

PLACEMENT BUREAU FOR X-RAY TECHNICIANS ESTABLISHED BY THE AMERICAN REGISTRY OF X-RAY TECHNICIANS

The American Registry of X-ray Technicians was established in 1921, under the joint sponsorship of the leading radiological societies, as a means of guiding the activities of the x-ray technicians, counteracting the tendencies toward lay-laboratories, and working generally in the interests of higher standards of technician training and the resulting technical improvement.

Since that time they have extended their facilities to include investigation and approval of technician training courses under desirable radiological directorship, and to the enforcement of adherence to ethical precepts on the part of x-ray technicians coming under the jurisdiction of the Registry Board. They now have undertaken the attempt to facilitate contact between well trained technicians and radiologists in need of such, by means of a Registry Placement Bureau.

Radiologists desiring assistants are invited to register with the Bureau, and contact will

be established by the Bureau with technicians meeting the specific requirements stated. Technicians will be classified according to their added abilities as nurses, laboratory technicians, etc., and will be given opportunities to apply for positions for which they are particularly fitted. It is hoped that a minimum of correspondence will result from the system that has been devised.

No fees are charged at any time in connection with the services of the Placement Bureau. Radiologists may register vacancies for x-ray technicians by writing to Alfred B. Greene, R.T., Executive Secretary, American Registry of X-ray Technicians, Oak Terrace, Minnesota.

OFFICERS AND COMMITTEES OF THE AMERICAN RADIUM SOCIETY

The following is a list of the Officers and Committees sent by the Secretary of the American Radium Society, for publication.

Officers

Frederick W. O'Brien, M.D., *President*, Boston.
Hayes E. Martin, M.D., *President-elect*, New York City.
LeRoy Sante, M.D., *First Vice-president*, St. Louis, Mo.
Leland R. Cowan, M.D., *Second Vice-president*, Salt Lake City, Utah.
Charles L. Martin, M.D., *Treasurer*, Dallas, Texas.
William E. Costolow, M.D., *Secretary*, Los Angeles, Cal.

Executive Committee

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CANCER (DIAGNOSIS)

Radiologic Aspects of Beginning Gastric Carcinoma. René A. Gutmann. Bull. et mém. Soc. de radiol. méd. de France, **27,** 15–17, January, 1939.

In addition to previously described radiographic appearances of early gastric cancer, *niche en plateau, niche triangulaire, niche encastrée,* the author calls attention to the niche in an arciform defect in the contour of the stomach. This is usually seen in the lesser curvature side. It may be very small, often too small to be seen radioscopically. The mucosal contour approaches the niche, then passes below it in an arc to rejoin the normal pattern. A slight difference of shading makes the lacunar image from which the niche emerges apparent. The niche does not project beyond the normal contour of the stomach.

This appearance is practically always indicative of early gastric cancer and, if left alone, will increase to form the large defect usually associated with cancer.

S.R. Beatty, M.D.

Bronchiogenic Carcinoma with Subcutaneous Metastases. Herman Charache. Am. Jour. Cancer, **37,** 431–434, November, 1939.

A case of carcinoma of the lung is reported, with two subcutaneous nodules which proved to be metastases. A review of the literature revealed that only 30 cases of subcutaneous metastases had been reported out of 1,063 cases of carcinoma of the lung. A careful examination of the skin might demonstrate more of this type of metastases.

Harold O. Peterson, M.D.

CANCER (THERAPY)

Several Years' Experience and Results Obtained with Near Distance Radiation in Carcinoma of the Skin. K. Henkel. Strahlentherapie, **66,** 108–118, 1939.

The author relates briefly his experience with the use of near distance radiation in the treatment of carcinoma of the skin. He believes that this method may not only replace radium therapy but is in certain instances superior to it. It is simple to apply, can be used upon ambulatory patients, and does not take much of the patient's time. Dosage is very accurate and because of the marked drop of radiant intensity during the first few centimeters very little normal tissue is unnecessarily irradiated. During the last four years Henkel has treated 101 cases of carcinoma of the skin, in which freedom from symptoms has been obtained in 96.4 per cent. Twenty-six of his patients are now under observation up to nearly four years. The cosmetic results obtainable are shown in a series of illustrations.

Ernst A. Pohle, M.D., Ph.D.

Results with Near Distance Radiation in Carcinoma of the Skin. F. John. Strahlentherapie, **66,** 132–142, 1939.

The author relates the experience of the skin clinic, of the University of Freiburg, with the use of near distance radiation in carcinoma of the skin. The technic was: 55 kv., 5 ma., 0.15 mm. Cu, 1.5 to 5 cm. F.S.D., single doses of approximately 600 r and total doses of from 5,000 to 6,000 r. The histories of 13 cases which were treated primarily by the method are presented in a table in condensed form and it is apparent that they responded well to the treatment. In a second table are briefly given the histories of five patients who had been irradiated previously and this influences the prognosis considerably. The author states that no definite rules can be given at present regarding the dose necessary for the individual case. The technic is still very flexible and subject to the experience of the individual radiologist.

Ernst A. Pohle, M.D., Ph.D.

Malignant Disease of the Vulva. Morris M. Datnow. Proc. Royal Soc. Med., **32,** 298–300, February, 1939.

This author describes the disease systematically. Quoted figures indicate that operation must not be expected to yield over 15 per cent five-year cures or 7 per cent absolute cures.

In the author's 35 cases the following facts are salient:

Average age, 63 years.	
Squamous-cell carcinoma	34
Spindle-cell carcinoma	1
Sites of lesion:	
Labium majus	18
Labium minus	8
Clitoris	7
Vestibule and urethra	2
Radical operation done	32
Radical operation and radium	3
Alive five and more years	10
Alive from one to five years	9
Known dead	10
Not traceable	3

The author employs radical resection, but hopes that improved technic will make more available the wider application and far lesser shock of radium treatment.

Because of the free lymphatic anastomosis between the mons veneris and the inguinal regions, recurrences are frequent in the mons; and superficial glands on either side become involved with the deep glands on the same side.

By radical operation the author generally aims at the removal of the superficial inguinal and femoral glands of both sides in one piece with the vulva.

Ray A. Carter, M.D.

The Treatment of Deep Infiltrating Carcinoma with Near Distance Radiation. H. Chaoul and K. Greineder. Strahlentherapie, **66,** 73–88, 1939.

The authors continue the report on their experience with near distance radiation. They are using 60 kv., 0.2 mm. Cu, 5 cm. F.S.D. and for fields of from 2 to 4

cm. diameter a daily dose of from 400 to 500 r. The total doses vary from 4,000 to 10,000 r. Healing after the peak doses takes longer, at least seven to eight weeks, and atrophy of the skin with telangiectasis usually follows. With this method, 264 cases of carcinoma of the skin were treated, 211 of which were superficial lesions and 53 infiltrating tumors. Of the first group, 98 per cent were primarily cured and of the second group 77.3 per cent. The respective figures for carcinoma of the lip were 94.2 and 80.7 per cent. For 118 patients three- to seven-year observation periods are available. Of 89 superficial lesions, 80 were cured, and 17 of 29 with infiltrating lesions. The respective figures for carcinoma of the lip were 16 out of 17 and 8 out of 13. The five-year survivals for both carcinoma of the skin and lip were 80.4 per cent for the superficial lesions and 73.6 per cent for the others. Encouraging results were also seen in the treatment of carcinoma of the thyroid, malignant melanoma, and carcinoma of the rectum. The authors conclude that in this method we have a very efficient means of treating cancer.

ERNST A. POHLE, M.D., Ph.D.

The Increase of the Effect of Roentgen Rays by Means of Local Short Wave Therapy. H. Korb. Strahlentherapie, **65,** 649–656, 1939.

The author treated a number of cases by a combination of short electric waves and x-rays. When possible, he gave the treatment simultaneously; a few patients were irradiated first and then given short wave diathermy. One case is interesting because the patient had two skin cancers, one in the supra-orbital region and one near the angle of the jaw. The former was given combined treatment and the latter x-ray therapy alone. It recurred and was cured by the combined treatment. Other cases include two of carcinoma of the vulva, two of the breast, and three of the cervix. Although this is a preliminary report, the author feels rather encouraged as to the future of this combined treatment.

ERNST A. POHLE, M.D., Ph.D.

THE CHEST

Dermoid Cyst of the Mediastinum with Rupture into the Pleural Cavity. Digby Wheeler. Canadian Med. Assn. Jour., **41,** 235–236, September, 1939.

A woman, 36 years of age, complained of shortness of breath, pain in the right chest, weakness, cough, expectoration, and loss of weight for one year. The right side of the chest bulged anteriorly, laterally, and posteriorly. X-ray examination revealed an opacity of the entire right chest, displacement of mediastinum to the left, and a large irregular mass extending from superior mediastinum into the left chest. Films made after drainage of the right pleural cavity showed the mass to be a mediastinal cyst.

The patient was not seen for six years, when she returned because of a miscarriage. The chest showed the same findings as on previous examinations. Symptoms were not so marked. Films made five months later, after aspirations, showed the cyst half filled with fluid and fluid in the right pleural cavity at the level of the fourth rib, midaxillary line.

Closed drainage was attempted, but was unsuccessful. Frequent aspirations followed. Patient died a few months later.

Postmortem findings showed the right pleural cavity communicating at the level of the second rib anteriorly, with a large mediastinal cyst that contained polypoid growths. Dirty, grayish material found in the pleural cavity was also present in the cyst. The author believes the cause of death was infection introduced by repeated aspirations. He also discusses the possibility of a happier outcome had aspirations been resorted to only for the purpose of relieving embarrassment.

This is the first reported case of rupture of a mediastinal cyst into the pleura which has been observed over so long a period of time.

M. L. CONNELLY, M.D.

THE CRANIUM

Radiology of the Third and Fourth Ventricles.—Part I. E. W. Twining. British Jour. Radiol., **12,** 385–418, July, 1939.

Radiography of the third ventricle is of great importance because it forms the pathway between the lateral and the fourth ventricle. The same criteria are applied to the diagnosis in the third and lateral ventricles, but because of the small size of the third, special technic is sometimes needed for its demonstration.

The anterior wall is formed by the lamina terminalis. There is a small indentation in its upper portion anterior to the foramen of Monro caused by the anterior commissure. The floor is crossed by the optic chiasma, in front of which is the supra-optic recess and behind is the infundibular recess. The anterior surface and the floor have the weakest walls so they tend to balloon forward and downward when the ventricle is dilated. The foramina of Monro lie at the anterior end of the junction of the roof and the lateral walls. The suprapineal recess is of variable size and projects backward from the posterior wall. The posterior wall also shows a small recess at the level of the pineal. There are three landmarks which enable the third ventricle to be picked up on the lateral roentgenograph, the foramina of Monro, the notch due to the chiasma usually immediately above the sella, and the pineal gland. The shadow of the third ventricle is usually faint.

The third ventricle may be seen adequately on routine views of the head in many cases, but if it is not well filled it may be necessary to use special technics to be sure whether or not the failure to fill is due to disease or simply inadequate filling. Routine views should include anteroposterior, supine, and erect (Towne), right and left lateral prone, and erect and occipital. Sometimes, to fill the ventricle completely the head must be greatly hyperextended, raising the shoulders and tilting the table, and sometimes it is necessary to tilt the head laterally.

The use of thorotrast is recommended because it gives better contrast, the complete ventricular system

can be outlined at one time, air-lock is avoided, the risks inseparable from the use of air are avoided. It is well tolerated at the time of injection but is followed for a few days by decided meningeal reaction. The subsequent course of the patient is satisfactory.

In hydrocephalus the third ventricle is enlarged in all diameters with downward bulging of the anterior portion. If hydrocephalus is due to a tumor or cyst in the posterior portion, the anterior end will show up clearly and give the impression that the ventricle is completely filled. The dilated ventricle in hydrocephalus may cause erosion of the anterior or posterior clinoids or both in the adult. This effect is rare in children.

The diagnosis of secondary sellar alteration due to hydrocephalus is usually suggested when the posterior clinoids show porosis and erosion without displacement. The distinction between the effects of third ventricle pressure and those due to cerebral tumors cannot usually be made from the roentgenograph.

The failure of the third ventricle to fill is of great significance, provided the method of filling is reliable.

Displacements are produced by supratentorial lesions and are usually lateral. Upward displacements are rare. Tumors below the ventricle usually bulge into the ventricle. Upward displacement may be caused by large, slow-growing, non-invasive tumors or congenital absence of the corpus callosum.

Tumors which produce filling defects are: suprasellar glioma and meningioma, tumors of the corpora mammillaria, meningioma of the olfactory groove, aneurysm, pituitary adenoma, tumors of the optic chiasm.

Curving of the septum pellucidum does not necessarily mean a tumor of the third ventricle blocking the foramen of Monro. This appearance may be due to sagging of the corpus callosum as the air gives less support than the cerebrospinal fluid. Uncontestable evidence of Monro block can be shown by attempting to equalize the fluid in the two ventricles at the fluoroscope. This can by no means be effected if the septum lucidum is intact. The decisive finding is given by the absence of the third ventricle shadow and by different fluid levels in the two anterior horns.

The floor of the third ventricle may be raised by a tumor arising in the tegmental region of the pons.

Tumors in the lateral ventricle in the neighborhood of the foramen of Monro may obstruct the foramen and cause non-filling of the third ventricle. Such tumors produce a circumscribed encroachment on the lateral ventricle.

SYDNEY J. HAWLEY, M.D.

GASTRO-INTESTINAL TRACT (DIAGNOSIS)

Chronic Superficial Gastritis: Correlation of Gastric Analysis and Clinical Study with Gastroscopic Examination. Joseph Bank and John F. Renshaw. Jour. Am. Med. Assn., **112,** 214–217, Jan. 21, 1939.

This study comprised a group of 50 patients with symptoms in the upper part of the abdomen in whom no abdominal disease other than gastritis was established. Each patient's complete history was taken and physical examination, roentgen study of the stomach, colon and gall bladder, examinations of the stool, sigmoidoscopic study, analysis of the blood and urine and bile drainages were done. Gastroscopic study was performed in the manner advocated by Schindler.

The frequency of foci of infection was outstanding; 43 (86 per cent) had definite foci of infection. Of these, 27 (54 per cent) had multiple foci. Foci of infection occurred as follows: infected teeth in 20 (pyorrhea in 12), diseased tonsils in 24, sinusitis in seven, chronic bronchitis in two, and giardiasis (intestinal infection) in one. The majority of their patients did not drink or smoke; neither of these agents was considered an important etiologic factor in any of their cases.

Prolonged duration of symptoms was a prominent feature in this group. This disorder appeared to begin in the third and fourth decades. There was a variation in the acidity from the normal value in 80 per cent of the series. Hyperacidity was present in 50 per cent and anacidity in 26 per cent. Gastric motility was delayed in 56 per cent of the series. A syndrome of duodenal ulcer was present in 26 per cent of the authors' 50 cases of chronic superficial gastritis. Loss of weight was a prominent feature, being noted in 46 per cent of the patients in the series.

CHARLES G. SUTHERLAND, M.D.

Megaduodenum and Gastromegaly. H. W. Gillespie. British Jour. Radiol., **12,** 221–224, April, 1939.

The history of a man, 30 years of age, who complained of flatulence, nausea, vomiting, and pain in the upper abdomen is given. X-ray examination revealed a dilated duodenum, with no apparent cause. This finding was confirmed at laparotomy. Subsequently, the stomach showed no dilatation.

Possible causes for this condition are discussed. The fact that symptoms did not appear until the patient was 30 years old probably rules out congenital origin. The author thinks a sympathetic-parasympathetic imbalance is the most likely cause.

SYDNEY J. HAWLEY, M.D.

Report of a Case of Stenosis of the Jejunum Caused by Tuberculosis. Luciano Nuzzo. Radiol. med., **26,** 664, 665, July, 1939.

The patient, a 55-year-old woman, gave a negative history, except for two daughters who had died of tuberculosis.

Her present illness began three months previous to operation. She complained of a feeling of weight in the epigastrium after eating, without pain or pyrosis, vomiting from four to five hours after meals. The vomitus was acid and contained what she had had for the meal. It never contained blood. Her appetite remained good.

Radiologic investigation of the gastro-intestinal tract revealed a marked stricture in the proximal portion of the jejunum with noticeable dilatation of the portion of

the intestine proximal to it, with well marked valvulæ conniventes. At operation the lesion was found to be located about 40 cm. distant from the ligament of Treitz.

Before resection the lesion was thought to be neoplastic.

ANTONIO MAYORAL, M.D.

GENITO-URINARY TRACT

Some Aspects of Renal Radiology. G. Vilvandré. British Jour. Radiol., **12,** 215–220, April, 1939.

The entire urinary tract should be examined in each case. The simpler procedures should be done first, as they assist in directing further examinations.

Particular attention must be given to that portion of the ureter which crosses the bony pelvis, as stones may easily be missed here. Anomalies of the urinary tract should be sought.

Various common urinary diseases are briefly discussed.

SYDNEY J. HAWLEY, M.D.

Hematuria and its Significance Following Automobile Accidents: Modern Means of Diagnosis and Treatment. Leo P. Dolan. Ohio St. Med. Jour., **35,** 825–827, August, 1939.

With the increasing number of automobile accidents, the occurrence of injury to the genito-urinary tract, especially rupture of the bladder, urethra, or kidney pelvis, is a serious complication of not infrequent incidence. The author presents two cases, one of a ruptured bladder complicating a fractured pelvis, and the other of a bilateral rupture of the kidney pelves. In his opinion and experience, intravenous urography is the only procedure which can definitely localize the site of injury. This is done without instrumentation from below and obviates the danger of introduction of infection into a presumably sterile urinary tract.

SIMON POLLACK, M.D.

GYNECOLOGY AND OBSTETRICS

Radiology in Pregnancy. J. B. Hartley. British Jour. Radiol., **12,** 193–214, April, 1939.

The author gives a summary of the method of making films and the technic of the Hooton method of pelvimetry. Tests of the accuracy of this method were made on a skeleton pelvis and it was found to be accurate to one-twelfth of an inch. (This method is similar to the Ball method, but gives only the transverse diameter and the true conjugate.)

In practically every case a fetus can be seen at the sixteenth week but in favorable cases it may be seen earlier if the mother is thin and well prepared and the proper technic is used. The absence of fetal structures after sixteen weeks does not necessarily mean that the patient is not pregnant; fetal movements may cause the shadows to be obscured.

The age of the fetus can be determined by cephalometry by studying the skeletal development of the fetus. The author does not give particulars for different ages. By the rate of skeletal development he has been able to predict the sex in five cases.

Fetal death may be diagnosed by several observations, the best of which is Spaulding's sign or overlapping of the cranial bones. Partial overlapping may be due to obesity, marked inclination of the pelvic inlet, occipito-posterior position, and prominent sacral promontory. The other signs of fetal death are failure of the fetus to enlarge in four weeks, and the "rolled-into-a-ball" appearance of the fetus.

Before examination, the pregnant patient should have the bowels cleaned out and the bladder emptied. For a complete examination the following things should be reported: presence or absence of a fetus, the number of feti, whether the fetus is dead or alive, relation of the presenting part to the pelvic canal, shape of the inlet and outlet, deformities of the pelvis, inclination of the inlet, fetal deformities, hydramnios, pelvimetry.

Cephalometry is best done in the prone position with a four-foot target distance. According to Robert's rule the biparietal diameter in inches is one-tenth of the duration of the pregnancy in weeks. One week should be subtracted in the case of fat women. No accurate statistics are available as to the reliability of this method.

The commonest causes of non-descent of the head at term are distention of bowels and bladder, occipito-posterior position, extreme inclination of the inlet, disproportion, abnormal presentation, placenta previa, intrapelvic tumor.

The following points are helpful in indicating that the child is overdue: size of the fetus, number of epiphyses visible, apparent disappearance of the sutures of the skull, and marked increase in the mineral content of the skull.

Abstracts of the predictions and the findings in 47 cases are given.

SYDNEY J. HAWLEY, M.D.

Radiation Therapy in Ovarian Dysfunctions. Franklin B. Bogart. South. Med. Jour., **32,** 708–711, July, 1939.

Care is advised in the selection of cases of ovarian dysfunction which are to be treated with irradiation. The present-day dosage of irradiation used is harmless to the patient and to the child of any future pregnancy. During pregnancy, radiation therapy to the pelvis should not be given.

For amenorrhea either irradiation of the pituitary alone or together with the ovaries has produced good results in 66 per cent of cases, good results being considered nearly normal menstruation lasting for six months. The dosage is small, the total dose reaching the ovaries not exceeding 10 per cent of a skin erythema.

Functional dysmenorrhea is treated similarly with 80 per cent satisfactory results. Menopausal syndrome is treated with 140 kilovolts, giving 175 r to each side of the head every three weeks for three treatments, with relief of symptoms in 73 per cent of cases. Headache is the symptom least amenable to treatment.

Functional uterine bleeding may be treated in young girls with the technic employed for amenorrhea. In older women, up to 250 r per field is given in divided doses over the pelvis. In cases in which menopause is desired, from 100 to 1,200 r per field is given. If immediate results are demanded, radium is applied within the uterus.

JOHN M. MILES, M.D.

HEART AND VASCULAR SYSTEM

Diverticulum Pericardi. Lewis Haas. Acta Radiol., **20,** 229–234, June 1939.

Diverticula of the pericardium may be congenital or acquired. The majority of pericardial diverticula belongs to the acquired group and may be defined as encapsulated pericarditic exudates at the site of adhesions.

However, it would be advisable to restrict the term "pericardial diverticulum" to the group of congenital origin only. Theoretically, these congenital diverticula may arise at any point of constitutional weakness in the pericardial layers. They are most frequently observed on the right heart contour at the site of the porta venosa. Frequently it has been asserted in the medical literature that no diverticula occur on the left side of the heart; this is not borne out by facts. The author, himself, presents two cases of pericardial diverticula, one of which was localized on the left heart silhouette.

ERNST A. SCHMIDT, M.D.

Calcification of the Aortic Valves. B. Moia and F. F. Batlle. Rev. argent. de cardiol., **6,** 108–115, May–June, 1939.

Calcifications of the aortic valves and ring exist more frequently than is generally believed; but because of the paucity of symptoms, the few and atypical clinical signs that are found in this condition, plus the great compensation power of the left ventricle, they are seldom diagnosed antemortem. Recent roentgenologic observations, however, have shown the possibility of demonstrating the existence of calcified valves in the living, bridging, thereby, a diagnostic lagoon in cardiology. The recent advancement is exceedingly important, not only in diagnosis, but also in prognosis, especially when the condition is found in subjects showing no symptoms.

The writers review the works of the leading pathologists in this field, and find that several theories have been advanced to explain the etiology, but they are inclined to agree with the German pathologist, Hampel, who, many years ago, expressed his opinion in these words: ". . . the differential diagnosis between pure valvular sclerosis and calcifications, sequelæ to acute endocarditis, still rests on a very weak basis."

As predisposing causes, age and sex are most important, as 75 per cent of calcified valves are found in persons over 50 years of age and males predominate in two to one proportion.

Clawson and his collaborator, studying autopsy material, classified the grades of aortic stenoses as follows: In 8.5 per cent there was no narrowing; in 20 per cent there was only a slight narrowing; in 26.5 per cent the narrowing was marked, and in 45 per cent it was quite well marked. In other words, in 75 per cent of the cases the stenosis was severe enough to give symptoms.

ANTONIO MAYORAL, M.D.

Visualization of the Chambers of the Heart, the Pulmonary Circulation, and the Great Blood Vessels in Heart Disease: Preliminary Observations. George P. Robb and Israel Steinberg. Am. Jour. Roentgenol. and Rad. Ther., **42,** 14–36, July, 1939.

Detailed structural visualization of the heart in six cases of rheumatic mitral stenosis and insufficiency has demonstrated the following points during life:

1. The left atrium enlarges toward the right side in the frontal view, forming the upper curve of the double contour, but it does not reach the left side except as the auricula which forms the eminence below the pulmonary arc.

2. The elevation and stenosis of the left bronchus appear to be caused by the enlarged left pulmonary veins and not the left atrium.

3. The accentuation of the hilar shadows and the increase in the lung markings are due to enlargement of both the arterial and venous components of the pulmonary circulation.

4. The main stem of the pulmonary artery is dilated but not lengthened, and it alone forms the pulmonary arc in the frontal view.

5. The conus arteriosus is dilated and lies farther to the left than normally, due to the elongation of the outflow tract of the right ventricle, but it does not enter into the formation of the pulmonary arc in the frontal view and plays only a minor rôle in the right oblique projection.

6. The enlargement of the right ventricle is due to dilatation and hypertrophy of the broad horizontal inflow tract and elongation of the narrow vertical outflow tract, with some widening of the conus arteriosus. In frontal view this chamber is broadened, forming three-fifths of the diaphragmatic contour.

7. The left ventricular cavity is moderately dilated and the wall thickened.

8. The superior vena cava, the right atrium, and the aorta remain normal in the absence of heart failure.

The visualization of the heart and the aorta in one case of syphilitic heart disease excluded gross anatomic change in the heart, and permitted the demonstration of disease of the intracardiac portion of the aorta which has previously been impossible during life. It thus made possible the determination of the exact size, shape, and location of the aneurysms and the thickness of the aortic wall.

Following the examination of the chambers of the heart and aorta in a case of hypertensive heart disease, it was found that the left ventricle was moderately dilated and hypertrophied, while the left atrium and the right chambers were normal in size and shape. The thoracic aorta itself was only slightly dilated but the

aortic loop was conspicuously tortuous and rounded in its course through the thorax. The innominate, the left common carotid, and the left subclavian arteries were dilated and tortuous, the tortuosity being sufficiently marked in the first one to cause "buckling." The left subclavian artery was shown to form the anterior boundary of the "aortic triangle."

In arteriosclerotic cardiovascular disease, of which one case is reported, there was noted that, (1) apparent enlargement of the heart was disproved by demonstration of the true cardiac borders; (2) the vascular nature of a rounded density near the left hilum which might have been called a calcified tubercle was shown; (3) the left atrium and ventricle were normal in size, shape, and location, and (4) the thoracic aorta exhibited a moderate degree of dilatation, elongation, and unfolding, with a moderate degree of thickening and calcification of the wall.

S. M. Atkins, M.D.

Radiologic Diagnosis of Valvular Calcifications. B. Moia and H. J. Acevedo. Rev. argent. de cardiol., **6**, 46–51, March–April, 1939.

The authors lament the little interest aroused by recent roentgenologic investigations demonstrating calcifications of the cardiac valves. These calcifications can not only be demonstrated but the valve affected may be localized. The importance of the work is evidenced by the frequency with which calcification appears in valves of patients afflicted with rheumatic endocarditis. It is true that in many cases these findings are only confirmatory; however, they assume tremendous importance when they are the only findings in cases clinically silent, and they also have a decided prognostic value.

Using a small aperture and from 70 to 75 kv. and 5 ma. in the fluoroscope, the observer may localize the left auriculoventricular junction which can be detected by its V-shape and lack of or diminished pulsation. The aortic and mitral valves normally are placed on an imaginary line drawn from the auriculoventricular junction following the auriculoventricular sulcus joining the horizontal midline at about 45°. The patient is examined in both the right and left oblique positions. When the valve is calcified, a dense shadow will be seen in an inverted comma, pendulous, swinging movement.

Once seen, it must be proven that the shadows are intracardiac calcifications, that they are in the valvular structure, and which is the valve calcified.

Extracardiac calcifications move in synchronism with the respiratory movement and not with the heart beat, and they can be projected away from the cardiac shadows, while valvular calcification, on the other hand, cannot be projected away from the cardiac shadows. They hold the same relative position to the cardiac border during deep respiration. Their excursion during a cardiac cycle should be greater than that of the cardiac borders, and the amplitude of the excursion should be increased in the right anterior oblique position.

So far, only intracardiac valvular calcifications have been identified. To select the valve affected, one must proceed to investigate further the position of the shadows. These, according to work done by Sosman and Wosika, should be above the imaginary line previously described. When they do not occupy this position, one should expect to find the aortic above and midway and the mitral below and to the left.

When the valvular ring is calcified it casts an O-, U-, or J-shaped shadow on the film. The technic used to show these calcifications is 1 meter distance, 200 ma., and 1/10 to 1/30 of a second.

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Studies of the Descending Thoracic Aorta in the Anteroposterior Position. Benjamin Enquin and Juan A. Aguirre. Rev. argent. de cardiol., **6**, 83–94, May–June, 1939.

If the descending thoracic aorta sometimes casts a good image on films taken in the standard posteroanterior position, it is evident that by using appropriate position and technic films can be made useful in studying this portion of the aorta. The essayists point out how little attention has been paid to this subject, believing this to be due to the fact that normally only the upper and that portion of the vessel to the left of the vertebræ can be shown in roentgenographs. They believe, however, that the roentgenologic observations of the descending portion of the thoracic aorta present positive diagnostic value. They consider the subject in four separate groups: technic; normal images; pathologic images, and clinical value.

Technic.—It is highly important that the anteroposterior position be used, the central rays coinciding with the sagittal plane of the thorax. For patients having normal sized hearts or showing only a slight enlargement, an overexposed teleroentgenogram in the above position is sufficient. In others, the following technic is recommended: place patient supine on a Bucky, use 1 meter distance, 150 ma., from 75 to 80 kv., and from 0.1 to 0.3 second.

Normal Image.—The normal aorta appears as a straight-line shadow on the left of the vertebral column, extending from the arch obliquely downward and inward until it blends with the shadow of the vertebral column. The right border is always confused with the column, and is, therefore, never visible. The image presents certain variations caused by age, habit, sex, and shape of the thorax. As a rule, the prominence to the left is more marked during childhood. Habitus and the configuration of the chest have a decided influence on the position of the shadow. In the asthenic, it is almost overlapped by the shadow of the vertebræ, while in the hyperthenics the shadow is well toward the left, at times showing a slight convexity. Diseases of the neighboring organs also influence the aortic shadow. In advanced mitral disease, with large auricle, the shadow is faint or absent, possibly because the rotation of the heart pulls the vessel mesially. Other conditions which will prevent satisfactory visualization of the descending aorta are: marked dilatation of the heart; thickened pleural bands; cold ab-

scesses, and deviations of the esophagus. The aorta is the mediastinal structure least liable to be misplaced when the mediastinum is displaced. This, however, may happen.

The following alterations of the image the authors consider to be pathologic: Increase of the visible area; tortuosities of the shadow; visibility of all the vessels, the borders running parallel to each other, and both borders visible

Clinically, the first and second alterations are found in syphilis and in atherosclerosed walls; the third may be found in high grades of arteriosclerosis or hypertension when the artery is elongated, while the fourth may be found with localized dilatation.

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HYSTEROGRAPHY

The Diagnostic Value of Hysterography. Albert Davis. Proc. Royal Soc. Med., **32,** 1211–1222, August, 1939.

To assess its clinical value, hysterography was employed in relevant cases for two years and the results were correlated with operative findings.

The Remmington Hobbs cannula, used for intrauterine glycerinization, was a simple and most satisfactory form of technic. A preliminary injection of morphine, 0.25 gr., is necessary. A general anesthesia is rarely needed, but if used a manometer is required to prevent over-distention. Lipiodol is the best contrast medium. An average amount of 10 c.c. is injected slowly under direct screen vision until the uterine outline is clear. Excessive pressure may cause a venous reflux.

Normal appearances vary with the distention, the period of the menstrual cycle, and the parity of the patient. Inadequate pressure may fail to fill an abnormally resistant segment. Serial films under differing pressures are required. A duodenal diaphragm is ideal for the purpose.

Contractions may be: (1) Multiple and small, appearing arrhythmatically on the three walls; (2) large, apical, slowly obliterating a cornua; (3) sudden spastic evacuation of the whole uterus. The last two result from considerable over-distention, often with tubal impermeability. The contractions occupy up to three or more minutes, and on the film are easily mistaken for filling defects in cases in which long-interval films are not utilized.

Hyperplasia is occasionally seen. The varying size of the uterus and of its projected outline must be considered.

Congenital abnormalities are not infrequent.

Fibroids usually give a smooth regular indentation. Sometimes there are small multiple irregularities, without the ragged defects seen in carcinoma. The position of a fibroid can usually be gauged and any gross displacement of the uterus seen.

Ovarian cysts usually displace the uterus without distorting its cavity.

Malignant disease of the body gives characteristic defects, ragged and irregular in the ulcerative form, large in the hypertrophic type. Hysterography may reveal unsuspected carcinoma of the body, associated with extra-uterine growths.

Endometrial hyperplasia, if extreme, may simulate carcinoma and require diagnostic curettage.

Retained products of fair size may present good films, but in cases of long standing and in which the fragments are small, careful study is required. Repeated films will eliminate simulation by contractions.

In conclusion it may be said that accurate interpretation and elimination of fallacies require constant practice and closest attention to details. The utility of the method is open to discussion. The author believes that it is not a routine weapon but has a definite, fairly narrowly limited use, chiefly in identifying carcinoma or retained products without the hazard of curettage, or in locating fibroids.

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